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American Heart Journal

VOL. 51 JUNE, 1956 No. 6

Original Communications

A STUDY OF THE VENOMOTOR TONE IN A SHORT INTACT VENOUS SEGMENT OF THE FOREARM OF MAN

G. E. BURCH, M.D., AND MAHDI MURTADHA, M.D.*

NEW ORLEANS, LA.

THE venous system of the circulation has not received as much attention as the arterial portion, not only because physiologic phenomena and diseases of the arteries are more readily discernible and a simple method for measuring arterial blood pressure is available, but also because the role of the venous system is considered by many physiologists and clinicians to be primarily passive.[1] That the veins actually are active is well established,[2-6] and that disturbances in venous function are important in health and disease of man is likewise accepted by many investigators.[7-21] Unfortunately, the complex nature of the entire cardiovascular system and its venous portion makes it difficult to study the intact veins of man without the complicating influences of the arterial and other segments of the circulatory system. The volume of the blood within and tissue fluids outside the veins, as well as the surrounding tissues, such as skeletal muscles, may vary so as to influence the state of the veins.

Because there are so many variables operating in the intact venous circulation, it was decided to attempt to isolate, with relatively little physiologic disturbance, segments of superficial veins on the forearm of intact man for study, to learn first if a satisfactory simple preparation could be obtained and then whether or not pertinent physiologic information could be gathered from such a preparation. Others in the past and some more recently have employed such a preparation.[22-24]

This report describes a simple method for isolating and studying an intact venous segment in the human forearm, as well as the influence of certain factors upon the venous tone in the isolated segment. The preparation and results should be useful in the study and understanding of the behavior of the extremely active veins of the arm.

These studies were aided in part by grants from the Public Health Service (H 143) and the Upjohn Company, Kalamazoo, Mich.

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MATERIALS AND METHODS

Twenty-one adult subjects, including patients from the Charity Hospital and students and physicians from the Medical School, were studied. Nineteen were men and two were women, their ages ranging from 22 to 69 years. All but five, with chronic severe congestive heart failure, were free from hemodynamic disturbances or cardiovascular disease; five were normal, five had peptic ulcer, two had diabetes, and there was one patient each with tension headache, indigestion, splenomegaly, and Raynaud's disease. Several subjects were investigated two or more times, and approximately 185 different segmental reactions were noted. Nine subjects were studied in the sitting position and the remainder lying supine in a hospital-type bed in a comfortable air-conditioned atmosphere.

Fig. 1.—Method of isolation of venous segment. Diagram of the apparatus employed, in place with the venous segment isolated. Wedges are screwed in position, isolating the venous segment. The forearm is steadied with large sandbags, and cross bars lay over small sandbags. The phlebomanometer is shown with the needle in position. During the experiment the meniscus in the glass adapter was watched through a magnifying glass with the aid of a fluorescent light, as shown.

The subjects selected had prominent superficial veins whose tributaries could be readily identified. Segments measuring 3 to 5 cm. in length and free from discernible tributaries and valves were isolated for study. Venous segments were selected by pressing upon a segment of vein with two fingers separated from each other by approximately 3 to 5 cm., so as to cause it to be distended with blood. Then, when pressure was placed upon the blood-filled segment, if no blood could be seen to be expressed from the segment, it was considered satisfac-

tory for study. Furthermore, satisfactory isolation was assured throughout the course of the experiments because the pressure within the segment remained considerably higher than the pressure in the adjacent venous system or that within the segment prior to its isolation. Whether or not small vasa venosum that permitted small leaks existed in the segments were not known, but this was not considered to be because of the prolonged period of greater pressure in the segment than in the adjoining veins. Short segments were studied to avoid difficulties from leakage into venous tributaries.

The subject's arm rested passively upon a table with the venous segment near the phlebostatic level,[25] except on six occasions when it was 5 to 60 mm. below, and in these instances the reading was corrected to the phlebostatic level. The arm was steadied with sand bags, which rested on the table against the arm (Fig. 1). Care was taken to avoid, by constricting clothing or position, ob-

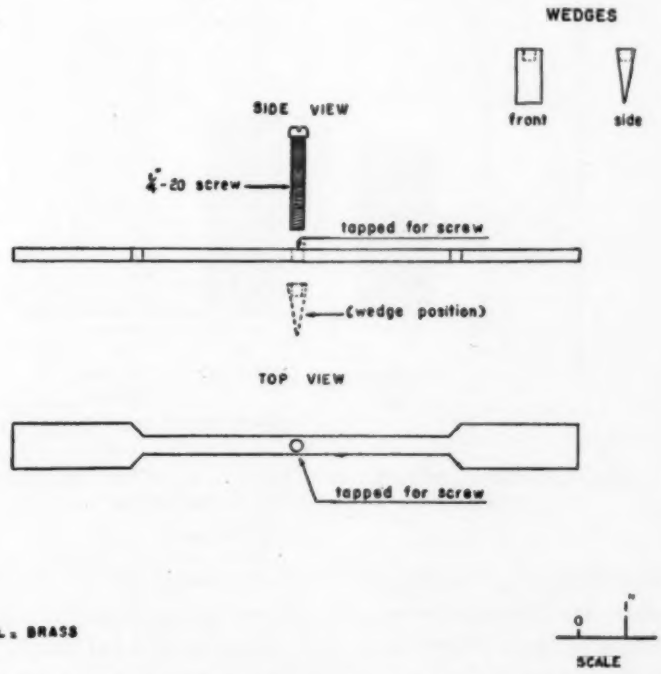

Fig. 2.—Diagram of brass wedges and cross bars used in isolating the venous segments of the forearm of man. The screw fits loosely into the depression in the superior face of the wedge. See text and Fig. 1 for further details.

struction to the mass of superficial veins on the volar surface of the forearm containing the segment isolated for study (Fig. 1). The subjects relaxed and were asked to cooperate as disinterestedly as possible in the experiments, and every attempt was made to avoid emotional or psychic disturbances unless such disturbances were intentionally introduced as a stimulus for study. All subjects were studied postprandially, and all but two were studied in midafternoon.

Detailed characteristics of the brass wedges and cross bars used to isolate the venous segments are shown in Figs. 1 and 2. The cross bars weighed 250 Gm. and the wedges 3.4 Gm. each. Their simple construction permitted ease in

isolation of any venous segment selected for study on the volar surface of the forearm, as well as space for work and observation of the veins.

The skin over the ends of the segment selected for study was marked off with ink. After the arm was placed in position with the sandbags, the segment was entered with the needle of the phlebomanometer,[20,26] and the pressure in the open segment was measured. The needle and adapter of the phlebomanometer were first rinsed with heparin solution (10 mg. per cubic centimeter), and then the 2 per cent solution of sodium citrate was drawn into the needle and adapter of the phlebomanometer as previously described.[20] Time was recorded (by stop watch) from the moment of entry of the needle into the isolated segment. The duration of the studies varied from two to forty-six minutes. After the pressure in the venous segment was recorded with blood

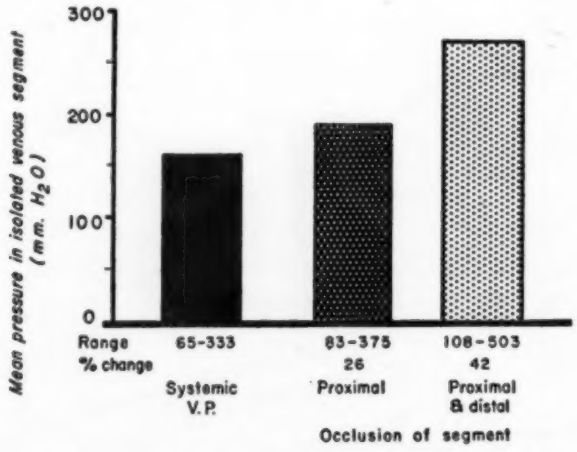

Fig. 3.—Influence of occlusion of the vein in isolation of a venous segment on the segmental venous pressure.

flowing freely through it, the proximal end was occluded with a metal wedge, and venous pressure was recorded and allowed to become stabilized. The distal end of the segment was then obstructed with the other metal wedge, venous pressure being recorded continuously and allowed to stabilize before the influence of any stimulus or other factor upon the segment was tested.

Reactions in the venous segments to the various factors introduced in the experiments were measured in terms of changes in pressure within the isolated segment. No new stimulus or factor was introduced until the pressure had again become stabilized at or near the level obtained after isolation of the segment. Because the segments were so small, relatively small variations in segmental luminal fluid volume could have altered intraluminal pressure, necessitating a constant segmental volume during the study. The pressure served as an index of the force, tension, or state of contraction[27-30] of the smooth muscle of the wall of the venous segment. The phlebomanometer satisfactorily maintained a constant volume in the isolated segment while the pressure within was being recorded. Precaution was taken to avoid adding fluid to, or removing

blood from, the isolated segment; that is, all venous pressure readings were obtained with the meniscus at the same point in the glass adapter. The meniscus was observed through a large 3X lens, and the field was well illuminated with fluorescent lamps. Insertion of the needle into the segment was made carefully to avoid kinking or distortion of the venous segment. The needle and adapter were loosely strapped in place with adhesive tape, and the apparatus and venous segment were not touched once measurement of venous pressure was begun.

RESULTS

The influence of each factor studied upon the pressure within the venous segment is described separately. In some preparations a flare of the type described by Lewis developed around the wedges and needle of the phlebomanometer, but this apparently did not influence the results.

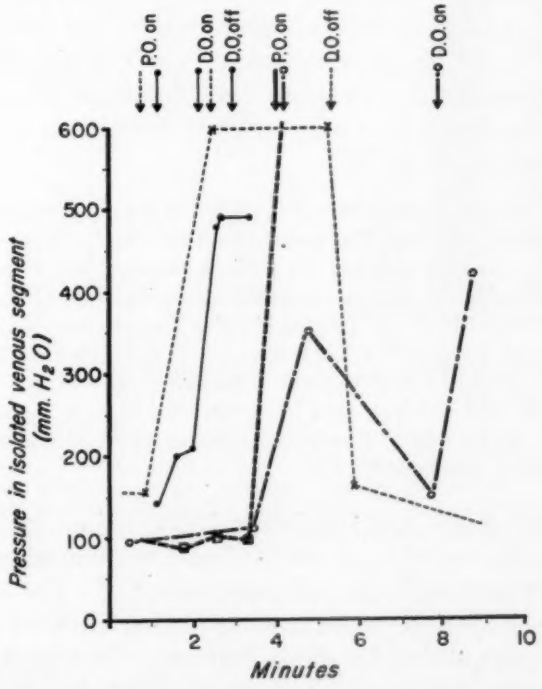

Fig. 4.—Influence of local venous spasm on segmental venous pressure. Curves of segmental venous pressure for four subjects in whom gross spasm developed in the wall of the segment. Respective arrows point to time when proximal occlusion (*P. O.*) and distal occlusion (*D. O.*) were imposed or removed. Note that spasm developed in one subject before distal occlusion was imposed; it was of such extent that the vein became umbilicated.

Occlusion of the Vein to Isolate the Venous Segment.—When the segment of vein selected for study was occluded proximally, the venous pressure usually increased slightly but rose to a greater extent when the distal end was also occluded (Fig. 3). There was a mean increase of 39 mm. of H_2O pressure with proximal occlusion (range 35 to 265 mm. H_2O) and another mean increase of 80 mm. of H_2O pressure with distal occlusion (range 50 to 267 mm. H_2O).

Comment.—The cause for this increase in pressure within the segment as it was obstructed for isolation is not known, but it appeared to be due in large part to compression of the surrounding tissues and venous segment by the edge

of the wedges. Visually detectable spasm occasionally developed in the venous segment, at which time the pressure usually rose to high levels. Conceivably, the trauma, although mild, may have irritated the smooth muscle locally to produce the gross spasm. Furthermore, mild spasm may have contributed to the elevation in pressure within the segment during its isolation.

Grossly Evident Local Smooth Muscle Spasm in the Wall of the Isolated Segment.—Local spasm sometimes developed in the venous segment, deforming it and at times even emptying it, apparently by forcing blood out at the occluded ends. When this occurred, the segment was not suitable for other studies. Range in pressure in the segment during spasm was from 420 to 600+ mm. H_2O (Fig. 4). The spasm lasted over thirty minutes occasionally, tending to disappear slowly in all instances, and was often manifested merely as local constricted rings that produced an hourglass deformity in the isolated segment.

Comment.—The cause for the spasm in the segment is not known. The needle puncture, undue movement of the phlebomanometer needle once it was in place, and the sharp edges of the occluding wedges may have been contributing factors.[31,32] The force of venous contraction was occasionally so great during local spasm that it produced a pressure over 600 mm. of H_2O. At times the venous segment was reduced to a narrow cord, which, on palpation, resembled an artery in its consistency.

Influences of the Mental State on Venous Tone.—When the subjects became interested in the studies or entered into conversation, the pressure within the isolated segment increased, the increase being especially pronounced when the subject engaged in a disturbing conversation or when he was approached with a syringe and needle for an injection (Fig. 5). The increase and subsequent decline with subsidence of the psychic disturbances were rapid. The mean rise in pressure was 93 mm. H_2O (range 25 to 175 mm. H_2O) for individual readings (Table I and Fig. 5). One subject exhibited a fall in segmental pressure of 37 mm. H_2O while "dozing" during the experiment. Venous tone was increased in the isolated segment in response to psychologic stimulation, even when the arterial and venous circulations were obstructed by application of a blood pressure cuff to the brachium (Fig. 14).

Comment.—The intense and consistent increase in venous tone in response to psychogenic stimulation is not surprising. Similar observations were described by Duggan[23] and by Page and associates.[24] In two of our subjects systemic venous pressure declined when they became frightened by the experiments and fainted. The explanation for these differences in response is not clear, but their existence must be remembered during any study of venous physiology. The "vasovagal syncope" described by Lewis,[33] which two of our subjects manifested with systemic or generalized venous hypotension, may have been related to sympathetic vasodilatation and parasympathetic (vagal) effects. Likewise, the subjects who had increased venous pressure in the isolated segment must have been manifesting more generalized sympathetic vasoconstrictor activity. The rheoplethysmogram[34] under similar circumstances showed great reduction in digital flow, apparently due to sympathetic vasoconstrictor activity. Digital rheoplethysmograms were never obtained during vasovagal syncope. It must be remembered, however, that either an increase or a decrease in venomotor activity can occur during psychogenic disturbances.[35] The decline in segmental pressure when the subjects dozed has been observed previously by Hooker[36] for systemic venous pressure. Halmagyi[15] did not find venous pressure to fall

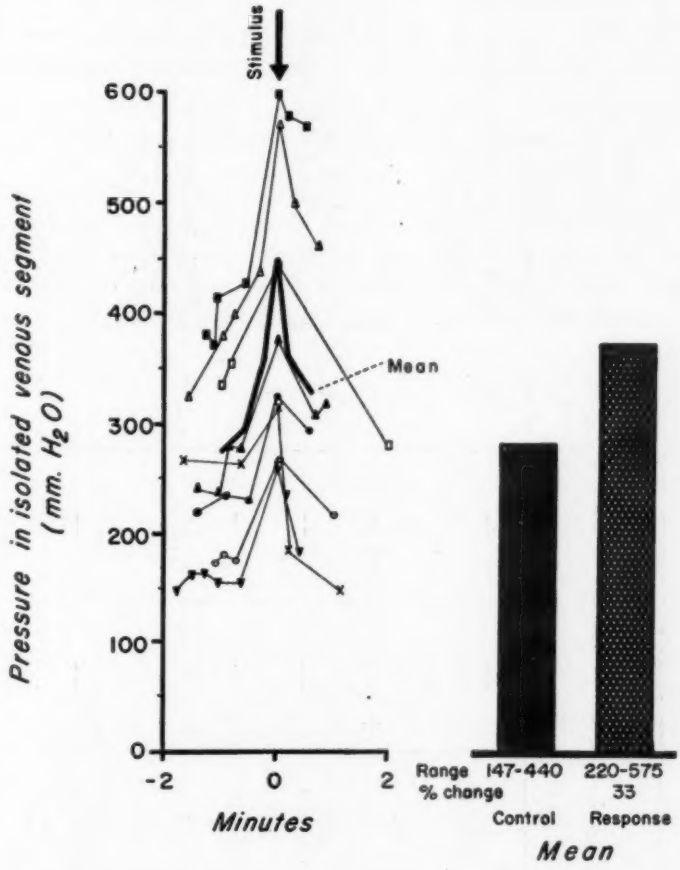

Fig. 5.—Time course of the individual pressures in the isolated segment and their mean in eight experiments, showing the influence of psychologic stimulation (arrow). Mental stimuli included thought, interest, fear, apprehension, and anger. The bar graph to the right of the figure represents the mean increase in pressure among fifteen observations following psychologic stimulation. In this and all similar figures to follow, the heavy continuous line represents the mean.

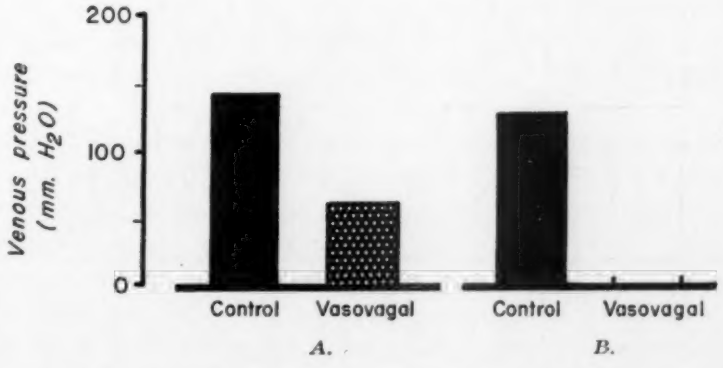

Fig. 6.—The systemic venous pressure fell in two subjects who spontaneously developed a vasovagal syndrome shortly after venipuncture. *A*, Patient 2. *B*, It was practically zero in Patient 7 at the height of his episode.

TABLE I. INFLUENCE OF VARIOUS FACTORS ON MEAN SEGMENTAL VENOUS PRESSURE

PROCEDURE	SEGMENTAL VENOUS PRESSURE						NO. OBSERVATIONS	COMMENT
	CONTROL*	RESULTANT*	RISE*		FALL*			
	(mm.H_2O)	(mm.H_2O)	(mm.H_2O)	(PER CENT)	(mm.H_2O)	(PER CENT)		
Deep inspiration	249 (123–473)	289 (142–553)	40 (5–160)	16 (4–113)	—	—	109	The ranges are for individual experiments and not for individual readings
Deep inspiration after procaine	203 (85–412)	199 (80–400)	—	—	4 (+3 to −12)	12 (+1 to −6)	27	
Psychogenic stimulation	274 (147–440)	367 (220–575)	93 (25–175)	34 (20–54)	—	—	15	One subject showed a drop with complete relaxation
Liver pressure	252 (197–314)	327 (281–402)	75 (40–105)	30 (13–44)	—	—	12	
Stroking	214 (161–239)	160 (127–162)	—	—	54 (33–76)	25 (11–48)	12	Four readings not included. One was zero and three showed a rise
Norepinephrine	179 (119–250)	600	421 (346–481)	234 (140–403)	—	—	3	
Hexamethonium	299 (255–335)	192 (135–245)	—	—	107 (10–198)	36 (4–59)	4	

*Range in parentheses.

with sleep in normal subjects but did observe a striking decline in the presence of congestive heart failure. Sleep induced by drugs may increase or decrease venous pressure, depending upon the actions of the drugs.[15]

Vasovagal Syncope and Systemic Venous Pressure.—It was most unusual to have had the opportunity to record the systemic pressure in a vein on the forearm of a subject in whom vasovagal syncope spontaneously developed from the psychic disturbance associated with these experiments. The two subjects who developed vasovagal syncope during measurement of the baseline venous pressure before the venous segment was isolated demonstrated progressive and fairly rapid decline in venous pressure as the syncopal syndrome advanced (Fig. 6). Recording of the venous pressure had to be discontinued so that the subjects could be made to lie on the floor of the laboratory before they became unconscious. These two subjects were sitting when the syndrome developed.

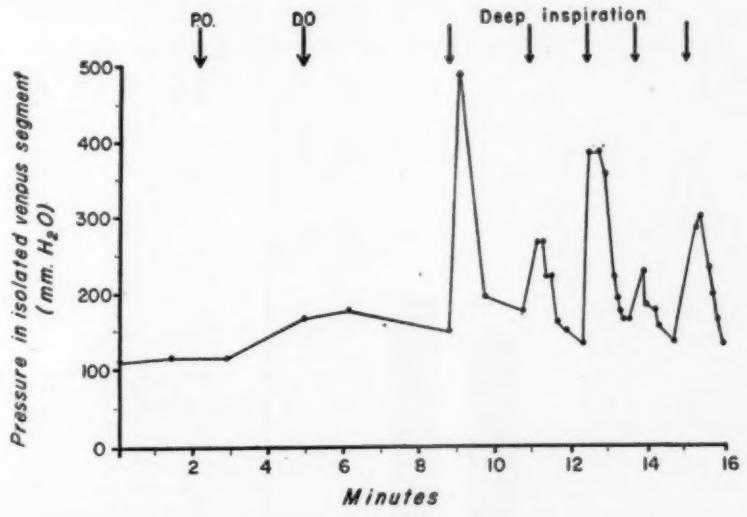

Fig. 7.—The time course of segmental venous pressure in a typical experiment, showing the influence of several deep inspirations.

Comment.—The decline in systemic venous pressure in the two subjects began before the investigators noted any clinical signs in the subjects of an impending syncopal episode, and the subjects did not make known their emotional disturbances if they were aware of any at the time. The progressive decline in venous pressure was the first indication of a systemic vasomotor disturbance. The venous pressure in one subject reached a zero level before recordings were discontinued.

Henstell and Gunther[37] found a decline in venous pressure in shock, whereas Robinson[38] and Ferris and associates[39] found it to remain unchanged in subjects with induced carotid sinus syncope. Wilkins and his co-workers[40] found venous pressure to decline in syncope, owing to nitrites. Apparently these are the first directly recorded observations of venous pressure changes in spontaneous syncopal episodes. The mechanism for the decline in venous pressure in this syncopal syndrome is not evident from these observations, but in the presence of venous hypotension veins do participate in the changes in circulatory dynamics associated with spontaneous psychogenically-induced syncope, which must be concerned with reflex pathways.

Deep Inspiration.—The patients were asked to inspire deeply and then exhale passively while the pressure in the isolated venous segment was being recorded. Deep inspiration resulted in an increase in pressure in the segment (Table I and Figs. 7 and 8) in all subjects, including those with chronic congestive heart failure, ranging from 5 to 160 mm. H_2O, with a mean of 40 mm. H_2O, or 16 per cent of 109 observations. At times there was a slight initial decline in pressure, followed by a progressive rise, then a decline to the original level. With repeated deep breaths there was a gradual decrease in the degree of response (Fig. 9) until it failed. This tachyphylactic type of reaction was consistent. The venoconstrictor response usually began within 2.5 to 3 seconds after deep inspiration; the segmental pressure rose gradually, reached a maximal value within the interval of several pulse cycles, and then returned gradually to the original pressure level within several additional pulse cycles. This response was interrupted by adequate infiltration of the subcutaneous area of the forearm proximal to the isolated segment with 2 to 5 c.c. of 1 per cent solution of procaine hydrochloride (Table I).

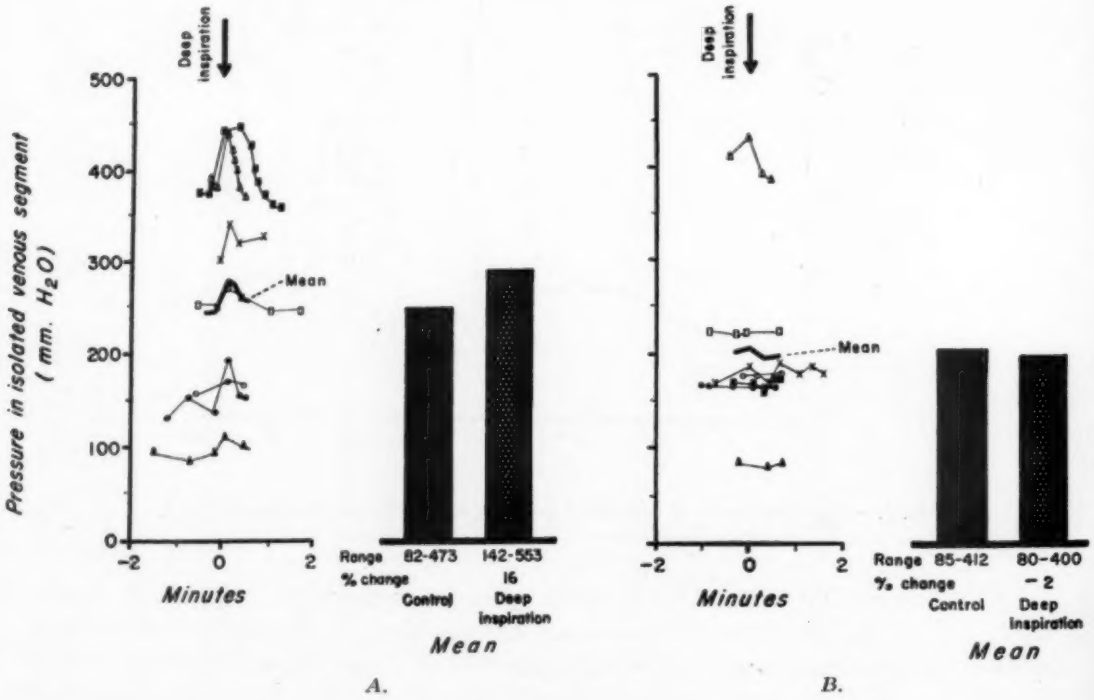

Fig. 8.—Illustration showing interruption, by procaine blocking of the innervation to the isolated segment, of the increase in segmental pressure produced by deep inspiration in three subjects. *A*, before and *B*, after administration of procaine.

Comment.—Deep inspiration is known to produce vasoconstriction in various peripheral vascular segments of the body of man,[41-53] a response that has been considered to be primarily arteriolar. For example, Mulinos and associates[45] did not find the veins to contract with deep inspiration. Hitzig[48] observed an increase in pressure in the jugular vein with deep inspiration in subjects with congestive heart failure, but not in normal subjects. Burton-Opitz[49] found the pressure in the jugular veins of dogs to decrease with inspiration and to increase with expiration, whereas with artificial respiration with the chest open, the reverse was true. Sewall[50] observed swelling of the superficial veins and increase

of pressure with deep inspiration. These latter two studies, however, apparently were concerned with the influence of the variation in intrathoracic pressure rather than reflex effects. In none of the reports found was there direct evidence of increase in venous tone.

More recent rheoplethysmographic studies have shown deep inspiration to result in constriction of the digital veins. These experiments demonstrate for the first time that the increase in vasomotor tone in response to deep inspiration involves the large superficial veins of the forearm of man and suggest strongly that other large veins also constrict following deep inspiration. Obviously, since vasoconstriction occurs in association with constriction of the smooth muscle in areas studied in which volume changes occurred, other portions of the vascular bed must dilate or distend to accommodate a shift in blood. The site of the respective changes and whether or not local action or passive venodilatation occurs are not known.

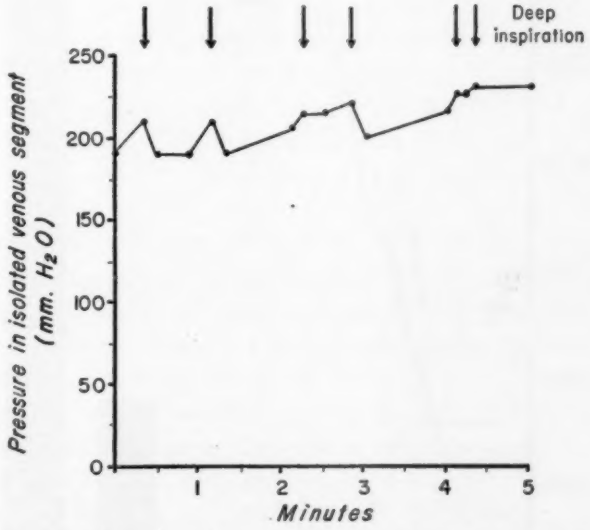

Fig. 9.—The time course of segmental venous pressure, showing the diminishing response of the venous segment to deep inspiration repeated at close intervals, suggesting "tachyphylaxis-type" phenomenon.

Stürup and associates[51] and Gilliat and co-workers[52] showed, for other vessels of the arterial side of the circulation, that this reflex is independent of cerebral lesions but dependent on the integrity of its pre- and postganglionic sympathetic nerve.

Bolton and Stürup[44] considered the different impulses for the reflex to arise from the thoracic wall, whereas Gilliat[53] thought it bore no relation to movement of the thoracic wall, but rather was related to the volume of air inspired. The study of Lieb and co-workers[54] suggests that an alveolar or bronchiolar stretch reflex may be responsible for the reaction. Its origin and nature of control and its function are yet to be elucidated. Since it has been demonstrated that superficial large and small veins participate in the reflex, the magnitude of the distribution is now evident.

Norepinephrine.—After the pressure had stabilized in the isolated segment in which sympathetic innervation had been previously interrupted by procaine, norepinephrine (0.05 c.c. of 1:100,000 aqueous solution) was introduced, by means of a 26-gauge needle and tuberculin syringe, into the isolated venous segment in three experiments. An equal volume of blood was withdrawn from the segment to maintain constant fluid volume in the segment. Within a few seconds after the norepinephrine was introduced, the pressure within the segment rose to 600 mm. Hg. (the limit of the recorder) or more in all instances (Table I and Fig. 10), blood was squeezed from the venous segment past the occluding wedges, and the segment was reduced in volume to a narrow rigid vessel. The segment was not suitable for further studies, and no effort was made to follow the return of pressure to its previous level, since the blood had been squeezed out of the segment.

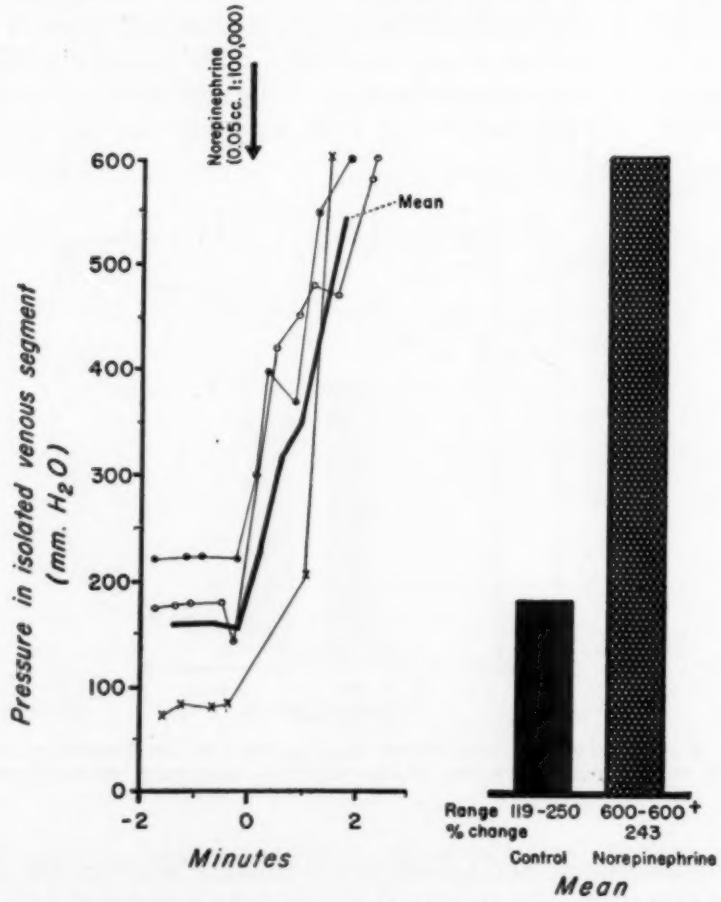

Fig. 10.—Influence of norephinephrine on segmental venous pressure, showing the sharp rise after its administration directly in the isolated segment of three subjects.

One contraction lasted as long as two hours. Occasionally, the contraction occurred initially at one end of the isolated segment, where the opening of the injected needle lay, and was intense enough to produce passive distention of the other portion of the segment not yet affected by the norepinephrine. As the drug diffused to all areas of the segment, the entire segment contracted, squeezing blood past the occluding wedges.

Comment.—The intense venoconstrictor effects of local intraluminal administration of norepinephrine on the isolated intact venous segment was expected and is consistent with the pharmacologic actions of the drug.[5,12,19,24,55-66]

It has long been known that epinephrine has a direct constricting action on veins, as was shown in studies on vein rings[55-59] and also in intact animals.[5,12,19,60-64] There have been only a few studies, however, of the action of norepinephrine on veins in man.[65,66] Fowler[65] presented evidence that it caused peripheral venous constriction and even suggested that this may be the primary factor in the arterial hypertension produced by norepinephrine. Von Euler[66] and Shadle and associates[67] noted that norepinephrine, in sufficient doses, might constrict the venules. Page and co-workers,[24] by injecting minute amounts of neosynephrine and epinephrine into the segment, caused it to contract but did not use norepinephrine. Contraction of the veins was intense and relatively prolonged, lasting two hours in one subject who apparently had slight extravasation of the drug around the segment of vein, as evidenced by an area of blanching.

This effect of norepinephrine on large veins must be considered during interpretation of its action on the circulation, including the heart. This drug, when administered intravenously, has also been found to produce constriction of the digital veins. Such widespread vasoconstricting action makes it difficult to interpret reactions to its use in physiologic studies, especially in man with disturbances in venous function, such as venous hypertension in congestive heart failure. Subjects with peripheral circulatory collapse and arterial hypotension, but venous hypertension, respond poorly to therapy with norepinephrine, or at least not as satisfactorily as subjects with simultaneous arterial and venous hypotension.

Certainly, the venous system of man responds to norepinephrine like the arterial system, with contraction of the smooth muscle within the vascular walls. Thus, norepinephrine apparently increases vasomotor tone throughout the vascular system with blood being displaced by actual vasoconstriction from some areas, probably peripheral, to others, probably central and possibly splanchnic, because of the effects of respective forces in different vascular segments governed by LaPlace's principles.[68] Local variations in tension in various portions of the cardiovascular system in association with a diffusely acting vasoconstricting factor need investigation. This study should be reduced, if possible, to the smallest contracting element in each smooth muscle fiber.

Hexamethonium.—Hexamethonium was administered into the median basilic vein of the opposite arm in doses of 5, 7.5, 10, and 12.5 mg. in four experiments on three subjects with chronic congestive heart failure while pressure was being recorded in the isolated venous segment of the opposite arm. The mean decline in the segmental pressure was 107 mm. H_2O, or 36 per cent (range 10 to 198 mm. H_2O) (Table I). As the pressure in the isolated segment declined (Fig. 11), there was a simultaneous decline in systemic venous pressure and arterial blood pressure. All subjects showed rather dramatic clinical improvement. No effort was made to follow the recovery of pressure in the isolated segment.

Comment.—Since hexamethonium acts mainly on the sympathetic nerve ganglia, the decrease in venous tone was effected reflexly in the isolated segment. These studies confirm earlier observations in this laboratory[69] that the drug acts upon the large veins of the forearm of man, as well as upon the arterial side of the circulation and the digital veins. Whether or not this action on large veins is general is not known. The decline in venous pressure noted in these subjects and others[70] is compatible with a generalized venodilator action of this drug.

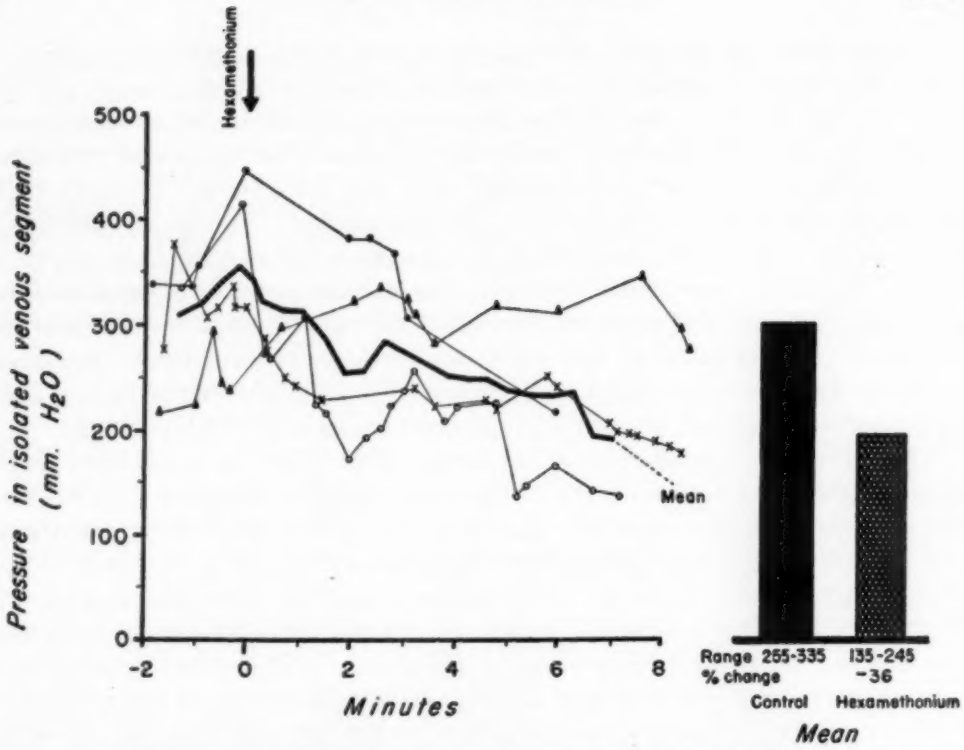

Fig. 11.—Time course of the isolated segmental venous pressure in four experiments on three subjects with chronic congestive heart failure, showing the influence of hexamethonium administered intravenously in the opposite arm.

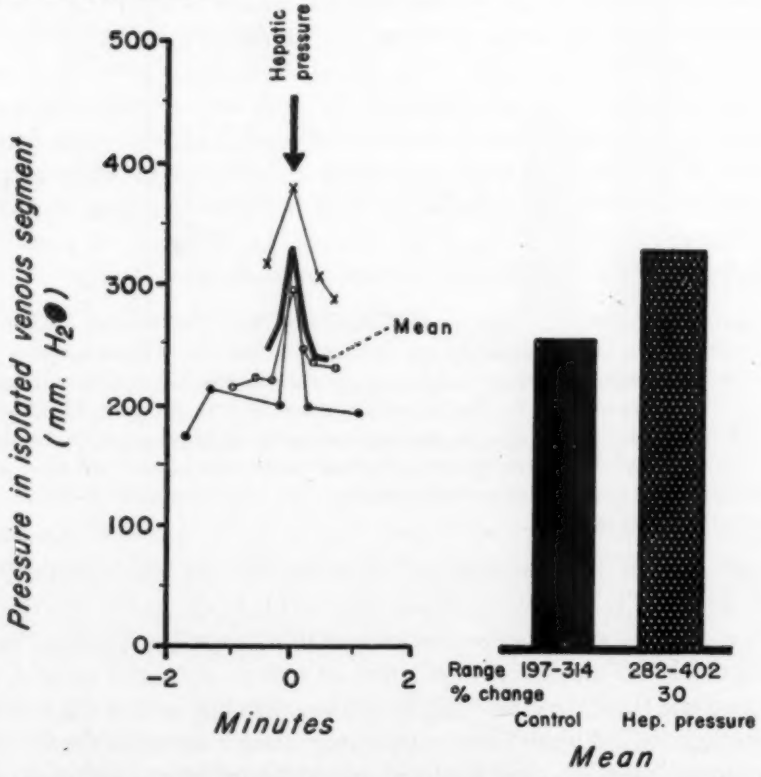

Fig. 12.—Influence of hepatojugular reflux on segmental venous pressure in three of four subjects studied.

The problems concerned with the distribution and shifting of blood within the vascular system are similar to that discussed for the effects of norepinephrine and deep inspiration. With a decrease in venomotor tone, local vasodilatation occurs, with shifting of blood into these areas from other vessels. On the other hand, if there is a generalized decrease in vascular tone throughout the arterial and venous sides of the circulation, the origin of the forces responsible for the volume shifts must again be related to differences in forces, according to the principles of LaPlace. Although the state of venous contraction decreases generally and pressure declines generally, force or tension within segments of the vascular system will vary with the shifting of blood within the vascular system, as determined by the relative magnitudes of the acting forces. These principles must be important not only in the venous circulation but also in that of the entire cardiovascular system.

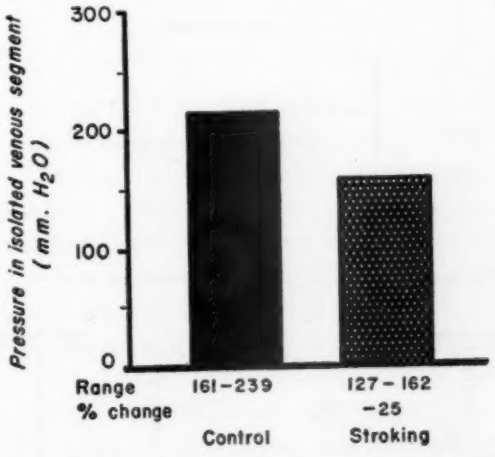

Fig. 13.—The mean decline in segmental pressure in twelve experiments produced by stroking gently over the isolated venous segment with the blunt, smooth end of a glass rod. There was an increase in pressure in only three instances and no change in one. Stroking the skin near the segment had no effect on segmental pressure.

Hepatojugular Reflux.—The hepatojugular reflux[20,48,71] was applied in four subjects with chronic congestive heart failure while pressure was recorded in the isolated venous segment of the volar surface of the forearm. Pressure over the liver resulted in an increase in pressure in the isolated venous segment in all four subjects (Table I and Fig. 12), with a mean increase of 75 mm. H_2O (range 40 to 105 mm. H_2O). The degree of increase could be made to vary with the amount of pressure applied over the hepatic region; with release of manual pressure, the pressure in the isolated segment declined rapidly to the former level.

Comment.—That the hepatojugular reflux caused a rise in pressure in the isolated venous segment, which was not continuous with the remainder of the general circulation, would suggest a sympathetic reflex stimulation of venous tone in the segment. This is a good possibility from existing knowledge of the physiologic state in chronic congestive heart failure.[14] Not only may function of the venosympathetic physiologic state be altered with generalized increase in venous tone, but also sympathetic activity may be increased.[14,15]

Stroking of the Skin Over the Isolated Venous Segment.—When the area of skin over the isolated venous segment was gently stroked with the smooth blunt end of a glass stirring rod, the venous tone of the segment usually declined, as evidenced by a decline in pressure within the segment (Table I and Fig. 13). The mean fall of 12 determinations was 54 mm. H_2O or 25 per cent (range 26 to 112 mm. H_2O). The occasional rise in pressure was the exception. Because the segment was closed, no volume change accompanied the pressure change.

Comment.—The response in venous tone with relaxation, produced by stroking the venous segment, is well known to clinicians who slap the skin over a vein to render it more prominent for venipuncture. The response in these experiments apparently was physical, with direct effect upon the smooth muscle in the venous wall, but it might also have been due to local chemical releases or local reflex action with secondary venodilatation.

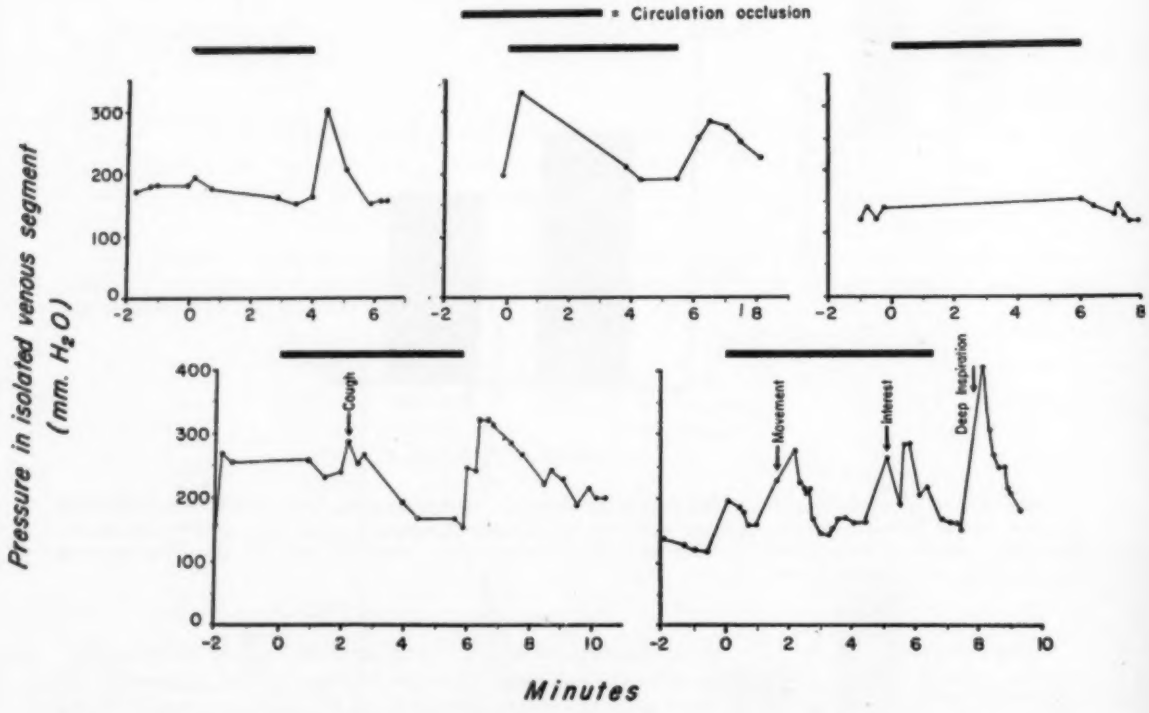

Fig. 14.—Influence of reactive hyperemia on segmental venous pressure. The time course of pressure in the isolated venous segment of five subjects in whom the circulation to the arm was interrupted for 4.25 to 6.5 minutes by means of inflating a blood pressure cuff applied to the brachium. Note the spontaneous fluctuations in segmental pressure during the period of circulatory arrest in the forearm and also the rise in segmental pressure with various stimuli applied during circulatory arrest. In three subjects a sharp, rapid increase in segmental pressure followed release of the circulation.

That the reaction was not dependent on sympathetic innervation was shown by Lewis,[72] who observed, by capillaroscopy, dilatation of the submaxillary venous network with stroking of the skin in a subject with sympathetic nerve degeneration following stellate ganglionectomy. The reason for an occasional instance of increased venous tone in these experiments is not known.

Reactive Hyperemia.—During the course of these experiments the circulation to the arm with the isolated venous segment was completely occluded at the brachium by inflation of a blood pressure cuff to a level greater than systolic arterial blood pressure for 4.25 to 6.5 minutes. During occlusion of arterial flow, the pressure in the isolated segment varied slightly without any definite directional trend. With release of the circulation and development of reactive hyperemia in the forearm and hand, the pressure within the isolated venous segment rose from 0 to 250 mm. H_2O (Fig. 14), returning to the level that prevailed prior to reactive hyperemia when the hyperemia subsided.

Comment.—The increase in pressure in the superficial venous segment of the forearm in response to reactive hyperemia in some subjects cannot be explained. The response could be reflex or chemical in origin, or both. Local changes in tissue pressure with engorgement of the adjacent vessels of the forearm may have pressed upon the venous segment to increase the pressure within, but because of the degree of rise in pressure, this could only have been a contributing factor.

GENERAL DISCUSSION

Maintenance of a constant volume within the segment while the varying pressure within was being measured was essential in these studies and was insured by use of the phlebomanometer. Because of the nature of the preparation and method of measurement, a rise or fall in intraluminal pressure in the segment must indicate an increase or decrease in tone of the smooth muscle in the wall of the venous segment. During the course of all the separate experiments, the isolated venous segments varied in prominence or diameter, and the venomotor tone and intraluminal pressure within also varied, reaching equally high levels for venous segments of various sizes, thus showing that venomotor tone can be high or low in veins in a state of either narrow or large bore. The possibility of variations in venous tone without change in luminal volume was suggested by Sewall[50] in 1906. The wall merely squeezed more tightly or less tightly upon the blood within. Only when the pressure within the segment rose to extremely high levels (600 mm. H_2O or more) was blood squeezed out of the segment past the points of obstruction.

The pressure within the isolated venous segments was also found to vary spontaneously. The oscillations in pressure were not regular but seemed to have a frequency resembling the alpha and beta waves described for the digital vascular bed.[73] The origin of these spontaneous variations in pressure was not determined but was probably related to sympathetic nerve activity. Hooker,[74] who described rhythmic changes in the degree of smooth muscle contraction in preparations of rings of the portal vein of rabbits, observed the venous tone to decrease upon exposure to CO_2. Franklin[4] noted rhythmic movements in rings made from the mesenteric vein of sheep. Haddy and associates[75] found spontaneous cyclic changes in pressure in small veins in the foot of dogs. Wharton Jones[76] and Nicoll[77] demonstrated, by capillaroscopy, rhythmic contractions in the veins of the bat wing. Kylin[78] and others[79] observed spontaneous variations in volume of the peripheral vascular structures that have been considered to be controlled by the sympathetic nervous system and to propel blood onward.[77]

Page and associates[24] published records that showed spontaneous variations in pressure, and these variations were also associated with reactions to a stimulus that varied venous pressure. Unfortunately, the portions of the records published are too short for adequate evaluation. These spontaneous contractions in veins may be related to peristaltic phenomena described for arteries by several physiologists in the past.[80] These somewhat rhythmic contractions were also noted in our experiments in the isolated venous segment during complete interruption of arterial and venous flow to the arm by inflation of a blood pressure cuff applied to the brachium (Fig. 14).

Because of the venomotor response of the isolated venous segment, and because of the many agents and stimuli used in these experiments, factors that increase or decrease arterial or arteriolar tone appear to have the same action on veins of man, as well as the venules, small veins, and A-V shunts in man's digits. This concept seems to be consistently supported by accumulation of more and more data on venomotor tone. Although it has been appreciated by physiologists for some time, it has not been widely emphasized.[1] If this is true, then, as stated earlier in this report, more information must be obtained concerning blood shifts in the venous system when venomotor tone increases or decreases throughout the venous system. Such moment-to-moment variations in systemic venous tone may be reflected in variations in right ventricular output.

These experiments also clearly show that the large veins, at least those of the forearm of man, react readily to many physical, chemical, and psychic stimuli. These reactions can be intense and associated with local and systemic variations in venous pressure, so that the veins should not be considered merely as tubes that passively conduct blood back to the heart, but rather as vessels that influence the state of the circulation. If the entire venous system participates in these reactions simultaneously, the influence of the changes in venomotor tone upon cardiac function and the circulatory state could be profound when the total volume of the venous system is considered. The essentially rhythmic variations in venous tone of the frequency of the alpha and beta deflections could be reflected in the entire circulation and rhythmic nature of venous return to the heart and, in turn, in cardiac output. Such influences might be of importance in disease or abnormal functional states. Integration of these venomotor tonal changes with the entire cardiac, arterial, and peripheral vascular functions and tonal states must be complex and necessary to maintain a normal circulatory state.

Finally, it should be noted that occasionally the response in the venous segment to deep inspiration was delayed, requiring as long as sixty seconds. This may have been the response of a type B vein described by Donegan.[12] Most of the venous segments, however, behaved like type A veins.

SUMMARY

By a simple method the venous pressure in an isolated intact segment of vein on the forearm of man has been studied. Since the volume of the segment was kept constant, changes in pressure in the segment were considered to reflect changes in "venous tone."

The influence of various phenomena on pressure in the segment was studied. Any form of mental excitation caused an increase in pressure in the segment. In one instance "natural sleep" was associated with a drop in pressure, probably a reflex mechanism. Deep inspiration produced a regular increase in segmental pressure, which was not obtainable when procaine was infiltrated proximal to the segment, suggesting a reflex mechanism. Injection of 0.05 c.c. of 1:100,000 solution of norepinephrine into the segment effected considerable increase in segmental pressure. Hexamethonium, injected intravenously in the opposite arm of patients with congestive heart failure, caused a fall in segmental pressure and subjective improvement in the patients; this was also probably a reflex mechanism. Pressure over the hepatic region in four patients with congestive heart failure was accompanied by a prominent rise of pressure in the segment, suggesting a reflex mechanism for the "hepatojugular reflux." Light stroking of the skin over the segment was associated with a fall in segmental pressure in most cases, a response that is considered to be local and mechanical.

Occlusion of the circulation to the forearm did not prevent spontaneous variations in segmental pressure or in the response to various stimuli, such as deep inspiration and mental excitation. Two subjects exhibited a sharp fall in systemic venous pressure while in the sitting position, heralding a vasovagal attack. Visible and palpable spasm of the segment, observed in a few subjects, increased segmental pressure remarkably. This was considered to be a local effect. Spontaneous variations in segmental pressure were frequent and resembled the alpha and beta deflections described for the digital vascular bed. They were considered probably to be related to sympathetic nerve activity.

Results suggest that venous tone is subject to the same forces that act on arterial or arteriolar tone. The veins are extremely important in both the normal and abnormal circulation, and further investigation of their behavior is encouraged.

SUMMARIO IN INTERLINGUA

Le pression venose in un isolate e intacte segmento de vena al antevracio human esseva studiate per medio de un simple methodo. Proque le volumine del segmento esseva mantenite a un nivello constante, il esseva possibile considerar alterationes de pression como reflexiones de alterationes in "tono venose."

Le influentia de varie phenomenos super le pression in le segmento esseva studiate. Omne forma de excitation mental causava un augmento del pression in le segmento. In un caso, somno natural esseva associate con un reduction de pression. Isto esseva probabilemente un mechanismo reflexe. Inspiration profunde produceva regularmente un augmento del pression segmental. Le facto que isto non esseva obtenibile post infiltration cis-segmental de procaina pare indicar que il se tractava de novo de un mechanismo reflexe. Le injection in le segmento de 0,05 cm^3 de norepinephrina in un solution de 1 a 100.000 effectuava un considerabile augmento del pression segmental. Hexamethonium, quando injicite intravenosemente in le bracio opposite de patientes con congestive disfallimento cardiac, causava un reduction del pression segmental e un melioration subjective del stato del patiente. Etiam in isto il se tractava probabilemente de un mechanismo reflexe. Pression supra le region hepatic esseva accompaniate in

quatro patientes con congestive disfallimento cardiac per prominente augmentos del pression in le segmento. Isto pareva indicar un mechanismo reflexe pro le refluxo hepatojugular. Leve massage del pelle supra le segmento esseva associate con un reduction del pression segmental in le majoritate del casos. Nos opina que iste responsa es local e mechanic.

Occlusion del circulation verso le antebracio non preveniva variationes spontanee in le pression segmental o in le responsa a varie stimulos, per exemplo inspiration profunde o excitation mental. Duo patientes exhibiva un abrupte reduction del systemic pression venose durante que illes se trovava in position sedente. Isto annunciava un attacco vosovagal. Visibile e palpabile spasmos del segmento, observate in un numero de subjectos, augmentava le pression segmental de maniera remarcabile. Isto esseva considerate per nos como un effecto local. Variationes spontanee del pression segmental esseva frequente e resimilava deflexiones alpha e beta del typo describite pro le rete vascular del digito. Nos opinava que iste variationes esseva probabilemente relationate a activitates sympathico-nervose.

Le resultatos pare indicar que le tono venose es exponite al mesme fortias que age etiam super le tono arterial e arteriolar. Le venas es extrememente importante in le circulation normal e anormal, e investigationes additional de lor conducta debe esser incoragiate.

REFERENCES

1. Landis, Eugene M., and Hortenstine, John C.: Functional Significance of Venous Blood Pressure, Physiol. Rev. **30**:1, 1950.
2. Hooker, D. R.: Evidence of Functional Activity on the Part of the Capillaries and Venules, Physiol. Rev. **1**:112, 1921.
3. Beaconsfield, Peter: Veins After Sympathectomy, Surgery **36**:771, 1954.
4. Franklin, K. J.: The Pharmacology of the Isolated Vein Ring, J. Pharmacol. & Exper. Therap. **26**:215, 1925.
5. Tryer, J. H.: The Effects of l-Adrenaline and dl-Noradrenaline on the Mean Right Atrial Pressure of Sheep With Intact Circulations, and on the Mean Inferior Vena Caval Pressure of Artificially Perfused Sheep, Quart. J. Exper. Physiol. **38**:161, 1953.
6. Bayliss, W. M., and Starling, Ernest H.: Observations on Venous Pressures and Their Relationship to Capillary Pressures, J. Physiol. **16**:159, 1894.
7. Henderson, Yandell: Tonus and the Venopressor Mechanism: The Clinical Physiology of a Major Mode of Death, Medicine **22**:223, 1943.
8. Henderson, Yandell: The Venopressor Mechanism, Science **95**:539, 1942.
9. McDowall, R. J. S.: The Nervous Control of the Blood Vessels, Physiol. Rev. **15**:98, 1935.
10. McDowall, R. J. S.: A Vago-Pressor Reflex, J. Physiol. **59**:41, 1924.
11. Heymans, Corneille: The Pressoreceptive Mechanisms for the Regulation of Heart Rate, Vasomotor Tone, Blood Pressure and Blood Supply, New England J. Med. **219**:147, 1938.
12. Donegan, J. F.: The Physiology of the Veins, J. Physiol. **55**:226, 1921.
13. Hooker, D. R.: The Veno-Pressor Mechanism, Am. J. Physiol. **46**:591, 1918.
14. Burch, G. E., and Ray, C. T.: A Consideration of the Mechanism of Congestive Heart Failure, Am. Heart J. **41**:918, 1951.
15. Halmagyi, D., Felkai, B., Ivanyi, J., and Hetenyi, G., Jr.: The Role of the Nervous System in the Maintenance of Venous Hypertension in Heart Failure, Brit. Heart J. **14**:101, 1952.
16. Alexander, Robert S.: Venomotor Tone in Hemorrhage and Shock, Circulation Research **3**:181, 1955.
17. Fleisch, Alfred: Venomotorenzentrum und Venenreflexe II. Mitteilung. Blutdruckzügler und Venenreflexe, Pflügers Arch. ges. Physiol. **226**:393, 1930.
18. Fleisch, Alfred: Venomotorenzentrum und Venenreflexe III. Mitteilung, Pflügers Arch. ges. Physiol. **228**:399, 1931.
19. Fleisch, Alfred: Die Wirkung von Histamin, Acetylcholin und Adrenalin auf die Venen, Pflügers Arch. ges Physiol. **228**:351, 1931.

20. Burch, G. E.: A Primer of Venous Pressure. Philadelphia, 1950, Lea & Febiger.
21. Franklin, K. J.: The Physiology and Pharmacology of Veins, Physiol. Rev. **8**:346, 1928.
22. Doupe, J., Krynauw, R. A., and Snodgrass, S. R.: Some Factors Influencing Venous Pressure in Man, J. Physiol. **92**:383, 1938.
23. Duggan, John J., Love, V. Logan, and Lyons, Richard H.: A Study of Reflex Venomotor Reactions in Man, Circulation **7**:869, 1953.
24. Page, E. B., Hickam, J. B., Sieker, H. O., McIntosh, H. D., and Pryor, W. W.: Reflex Venomotor Activity in Normal Persons and in Patients With Postural Hypotension, Circulation **11**:262, 1955.
25. Winsor, Travis, and Burch, G. E.: Use of Phlebomanometer: Normal Venous Pressure Values and a Study of Certain Clinical Aspects of Venous Hypertension in Man, Am. Heart J. **41**:387, 1946.
26. Burch, G. E., and Winsor, Travis: The Phlebomanometer: A New Apparatus for Direct Measurement of Venous Pressure in Large and Small Veins, J.A.M.A. **123**:91, 1943.
27. Kesson, J. E.: The Elasticity of the Hollow Viscera, Quart. J. Exper. Physiol. **6**:355, 1913.
28. Remington, J. W., and Alexander, R. S.: Stretch Behavior of the Bladder as an Approach to Vascular Distensibility, Am. J. Physiol. **181**:240, 1955.
29. Tang, Pei-Chin, and Ruch, T. C.: Non-Neurogenic Basis of Bladder Tonus, Am. J. Physiol. **181**:249, 1955.
30. Evans, C. Lovatt: Physiology of Plain Muscle, Physiol. Rev. **6**:358, 1926.
31. Robertson, Rocke H.: Vasospasm, Bull. Vancouver M. A. **24**:92, 1947.
32. Pereira, A. DeSousa: The Innervation of the Veins, Surgery **19**:731, 1946.
33. Lewis, Thomas: A Lecture on Vasovagal Syncope and the Carotid Sinus Mechanism, Brit. M. J. **1**:873, 1932.
34. Burch, G. E.: Digital Plethysmography, New York, 1954, Grune & Stratton, Inc.
35. Vincent, Sewale, and Thompson, J. H.: Further Observations on the Vasomotor Reflexes and Associated Phenomena, J. Physiol. **65**:327, 1928.
36. Hooker, D. R.: Observations on the Venous Blood Pressure in Man, Am. J. Physiol. **35**:73, 1914.
37. Henstell, Henry H., and Gunther, Louis: Studies of Plasma Volume in the Human Being; Comparative Results of Reduction of Plasma Volume, Intramuscular Pressure and Venous Pressure in Surgical Shock, Am. J. M. Sc. **209**:187, 1945.
38. Robinson, Leon J.: Venous Blood Pressure Measurements During Syncope Caused by a Hyperirritable Carotid Sinus Reflex, Am. J. M. Sc. **197**:100, 1939.
39. Ferris, Eugene B., Jr., Capps, Richard B., and Weiss, Soma: Carotid Sinus Syncope and Its Bearing on the Mechanism of the Unconscious State and Convulsions. A Study of 32 Additional Cases, Medicine **14**:377, 1935.
40. Wilkins, Robert W., Weiss, Soma, and Haynes, Florence W.: The Effect of Epinephrine in Circulatory Collapse Induced by Sodium Nitrite, J. Clin. Invest. **17**:41, 1938.
41. Binet, Alfred, and Sollier, Paul: Recherches sur le pouls cérébral dans ses rapports avec les attitudes du corps, la respiration et les actes psychiques, Arch. de physiol. norm. et path., Par. **7**:719, 1895.
42. Uhlenbruck, P.: Quoted by Bolton et al.[44]
43. Goetz, R. H.: Der Fingerplethysmograph als Mittel zur Untersuchung der Regulationsmechanismen in peripheren Gefässgebieten, Pflügers Arch. ges. Physiol. **235**:271, 1935.
44. Bolton, B., Carmichael, E. A., and Stürup, G.: Vaso-constriction Following Deep Inspiration, J. Physiol. **86**:83, 1936.
45. Mulinos, Michael G., and Shulman, Israel: Vaso-constriction in the Hand From a Deep Inspiration, Am. J. Physiol. **125**:310, 1939.
46. Wilkins, R. W., Doupe, J., and Newman, H. W.: The Rate of Blood Flow in Normal Fingers, Clin. Sc. **3**:403, 1938.
47. Hertzman, Alrick B., and Dillon, John B.: Selective Vascular Reaction Patterns in the Nasal Septum and Skin of the Extremities and Head, Am. J. Physiol. **127**:671, 1939.
48. Hitzig, William M.: On Mechanisms of Inspiratory Filling of the Cervical Veins and Pulses Paradoxus in Venous Hypertension, J. Mt. Sinai Hosp. **8**:625, 1941.
49. Burton-Opitz, Russell: Venous Pressures, Am. J. Physiol. **9**:198, 1903.
50. Sewall, Henry: Experiments on Venous Blood Pressure and Its Relations to Arterial Pressure in Man, J.A.M.A. **47**:1279, 1906.
51. Stürup, G., Bolton, B., Williams, D. J., and Carmichael, E. Arnold: Vasomotor Responses in Hemiplegic Patients, Brain **58**:456, 1935.
52. Gilliatt, R. W., Guttman, L., and Whitteridge, D.: Inspiratory Vaso-constriction in Patients After Spinal Injuries, J. Physiol. **107**:67, 1948.
53. Gilliatt, R. W.: Vaso-constriction in the Finger After Deep Inspiration, J. Physiol. **107**:76, 1948.
54. Lieb, Charles C., Mulinos, Michael G., and Taylor, Helen L.: Studies on Vaso-motor Reflexes. Vasoconstriction From a Deep Inspiration of Air, Proc. Soc. Exper. Biol. & Med. **34**:89, 1936.

55. Gunn, J. A., and Chavasse, F. B.: The Action of Adrenine on Veins, Proc. Roy. Soc., London, s. B. **86**:192, 1913.
56. Crawford, Albert C., and Twombly, Margret M.: Notes on the Response of Veins to Epinephrine, New York M. J. **98**:327, 1913.
57. Edmunds, Charles W.: Some Vasomotor Reactions of the Liver With Special Reference to the Presence of Vasomotor Nerves to the Portal Vein, J. Pharmacol. & Exper. Therap. **6**:569, 1915.
58. Maloff, G.: Pharmakologische Versuche an isolierten Venen des Menchen, Pflügers Arch. ges. Physiol. **229**:38, 1931.
59. Campbell, J. Argyll: The Effects of Certain Animal Extracts Upon the Blood Vessels, Quart. J. Exper. Physiol. **4**:1, 1911.
60. Connet, Helene: The Effect of Adrenalin on Venous Blood Pressure, Am. J. Physiol. **54**:96, 1920.
61. Hooker, D. R.: The Functional Activity of the Capillaries and Venules, Am. J. Physiol. **54**:30, 1920.
62. Hartman, Frank A., Evans, Jay I., and Walker, H. G.: Control of Capillaries of Skeletal Muscle, Am. J. Physiol. **90**:668, 1929.
63. Burton-Opitz, Russell: The Vascularity of the Liver. XI. The Motor Reaction in the Portal Radicles of the Liver, Quart. J. Exper. Physiol. **7**:57, 1914.
64. Fleisch, Alfred: Venomotorenzentrum und Venenreflexe. I. Mitteilung, Pflügers Arch. ges. Physiol. **225**:26, 1930.
65. Fowler, N. O., Westcott, R. M., Scott, R. C., and McGuire, J.: The Effect of Nor-Epinephrine Upon Pulmonary Arteriolar Resistance in Man, J. Clin. Invest. **30**:517, 1951.
66. von Euler, U. S.: Distribution and Possible Physiologic Functions of Epinephrine and Nor-epinephrine. In Transactions of Second Conference on Shock and Circulatory Homeostasis, Oct. 19-21, 1952, New York, 1953, Josiah Macy, Jr. Foundation, pp. 162-259.
67. Shadle, Oscar W., Moore, James C., and Billig, Donal M.: Effect of 1-Arterenol Infusion on "Central Blood Volume" in the Dog, Circulation Research **3**:385, 1955.
68. Burton, Alan C.: Physical Equilibrium of Small Blood Vessels, Am. J. Physiol. **164**:319, 1951.
69. Burch, G. E.: A Method for Measuring Venous Tone in Digital Veins of Intact Man, Arch. Int. Med. **94**:724, 1954.
70. Burch, R. R.: The Effects of Intravenous Hexamethonium on Venous Pressure of Normotensive and Hypertensive Patients With and Without Congestive Heart Failure, Circulation **11**:271, 1955.
71. Burch, G. E., and Ray, C. T.: Mechanism of the Hepatojugular Reflux Test in Congestive Heart Failure, Am. Heart J. **48**:373, 1954.
72. Lewis, T., and Landis, E. M.: Some Physiological Effects of Sympathetic Ganglionectomy in the Human Being and Its Effects in a Case of Raynaud's Malady, Heart **15**:151, 1930.
73. Burch, G. E., Cohn, A. E., and Neumann, C.: A Study by Quantitative Methods of the Spontaneous Variations in Volume of the Finger Tip, Toe Tip, and Postero-superior Portion of the Pinna of Resting Normal White Adults, Am. J. Physiol. **136**:433, 1942.
74. Hooker, D. R.: The Effect of Carbon Dioxide and of Oxygen Upon Muscular Tone in the Blood Vessels and Alimentary Canal, Am. J. Physiol. **31**:47, 1912.
75. Haddy, F. J., Richards, A. G., Alden, J. L., and Visscher, M. B.: Small Vein and Artery Pressure in Normal and Edematous Extremities of Dogs Under Local and General Anesthesia, Am. J. Physiol. **176**:355, 1954.
76. Jones, T. Wharton: Discovery That the Veins of the Bat's Wing (Which Are Furnished With Valves) Are Endowed With Rhythmical Contractility, and That the Onward Flow of Blood Is Accelerated by Each Contraction, Phil. Tr. Roy. Soc. Part 1, 131, 1852.
77. Nicoll, Paul A., and Webb, Richard L.: Blood Circulation in the Subcutaneous Tissue of the Living Bat's Wing, Ann. New York Acad. Sc. **46**:697, 1946.
78. Kylin, E.: Peristalsis of Capillaries, Klin. Wchnschr. **2**:14, 1923.
79. Chambers, Robert, and Zweifach, B. W.: Functional Activity of the Blood Capillary Bed, With Special Reference to Visceral Tissue, Ann. New York Acad. Sc. **46**:683, 1946.
80. Cornwall, Edward E.: Present Status of the Theory of Vascular Peristalsis as a Motive Factor in the Circulation, M. Times & Long Island M. J. **61**:268, 1933.

A METHOD FOR THE STUDY OF THE FORM OF THE HIGH FREQUENCY UNDAMPED BALLISTOCARDIOGRAM

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THE ballistocardiogram is a graphic recording of the movements of the body which result from the impact of the heart and the movement of the blood during each heart beat. Up to the present time a number of different types of ballistocardiographs have been described, and the form of the tracing which is obtained depends upon the system which is used.[1-4] This study deals with an objective method for the analysis of the pattern obtained with the high frequency undamped ballistocardiograph developed by Starr and his associates to record displacement of the body.[1] A number of relatively characteristic changes have been noted in the BCG of patients with various types of heart disease. These alterations in the form of the BCG have, however, only been described in qualitative terms in the majority of instances.

In the present study an attempt was made to correlate the I. J. segment with the G.H., H.I., J.K., K.L., L.M., and M.N. segments of the BCG. It was found that these correlations were sufficiently good in a series of normal subjects to allow standards to be established, by means of regression equations, for the prediction of the size of the latter segments from the size of the I.J. segment. Such an analysis provided an objective, though empiric, method for assessing the disturbances in the ballistocardiographic patterns which may result from heart disease.

MATERIAL AND METHODS

High frequency undamped ballistocardiograms were recorded on 131 normal men and 75 normal women. Their ages ranged between 18 and 35 years. The ballistocardiograms were recorded after the subjects had been at rest on the ballistocardiograph for at least fifteen minutes and more than two hours had elapsed since their last meal.

The ballistocardiograph was similar in principle to that described by Starr and associates and had a frequency response of 10 cycles per second when loaded with a dead weight of 150 pounds.[1] The movement of the table was amplified by means of a linear differential transformer and was recorded simultaneously with an electrocardiogram, a phlebogram, and a tracing of thoracic movements on a multichannel direct-writing oscillograph.

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TABLE I

AVERAGE OF 10 CONSECUTIVE COMPLEXES — REGRESSION EQUATIONS	SD	r	LEVEL OF SIGNIFICANCE FOR r: P
1. *50 normal male subjects*			
G.H. = 0.150 IJ + 0.03	1.36	+0.52	0.001
H.I. = 0.533 IJ − 1.190	1.37	+0.88	0.001
J.K. = 0.923 IJ + 1.457	1.67	+0.90	0.001
K.L. = 0.444 IJ − 0.638	2.13	+0.70	0.001
L.M. = 0.278 IJ − 0.347	2.06	+0.54	0.001
M.N. = 0.371 IJ + 1.040	1.84	+0.69	0.001
2. *75 normal female subjects*			
G.H. = 0.113 IJ + 0.359	0.80	+0.28	.05
H.I. = 0.420 IJ − 0.020	0.75	+0.75	0.001
J.K. = 0.867 IJ + 2.261	1.38	+0.78	0.001
K.L. = 0.379 IJ + 0.672	1.62	+0.43	0.001
L.M. = 0.305 IJ − 0.249	0.96	+0.54	0.001
M.N. = 0.384 IJ + 0.808	1.02	+0.60	0.001
G.H. = 0.360 HI − 0.030	0.68	+0.52	0.001
3. *Normal male and female subjects together*			
G.H. = 0.14 IJ + 0.20	0.34	+0.55	0.001
H.I. = 0.56 IJ − 1.60	1.04	+0.91	0.001
J.K. = 0.90 IJ + 1.90	2.02	+0.92	0.001
K.L. = 0.38 IJ + 0.60	1.76	+0.71	0.001
L.M. = 0.23 IJ + 0.57	1.30	+0.65	0.001
M.N. = 0.24 IJ + 2.74	1.89	+0.51	0.001
F.G. + G.H. = 1.51 IJ − 8.58	3.84	+0.59	0.001

INSPIRATORY AND EXPIRATORY COMPLEXES — REGRESSION EQUATIONS	SD	r	LEVEL OF SIGNIFICANCE FOR r: P
SINGLE LARGEST INSPIRATORY COMPLEX			
4.(a) *131 normal male subjects*			
H.I. = 0.468 IJ + 0.686	1.60	+0.80	0.001
J.K. = 0.848 IJ + 1.690	2.76	+0.81	0.001
K.L. = 0.459 IJ − 1.396	2.57	+0.64	0.001
L.M. = 0.299 IJ + 0.470	2.05	+0.56	0.001
M.N. = 0.293 IJ + 3.228	2.07	+0.49	0.001
(b) *60 normal female subjects*			
H.I. = 0.452 IJ + 0.016	1.10	+0.77	0.001
J.K. = 0.771 IJ + 3.213	1.47	+0.84	0.001
K.L. = 0.302 IJ + 1.639	1.71	+0.46	0.001
L.M. = 0.285 IJ + 0.431	1.20	+0.57	0.001
M.N. = 0.259 IJ + 2.722	1.87	+0.37	0.001
SINGLE SMALLEST EXPIRATORY COMPLEX			
5.(a) *131 normal male subjects*			
H.I. = 0.434 IJ + 1.112	1.53	+0.72	0.001
J.K. = 0.785 IJ + 1.929	2.16	+0.80	0.001
K.L. = 0.342 IJ − 0.110	2.00	+0.53	0.001
L.M. = 0.286 IJ + 0.215	1.82	+0.50	0.001
M.N. = 0.356 IJ + 1.968	2.07	+0.54	0.001
(b) *60 normal female subjects*			
H.I. = 0.428 IJ + 0.362	0.94	+0.74	0.001
J.K. = 0.856 IJ + 1.714	1.37	+0.83	0.001
K.L. = 0.252 IJ + 1.727	1.40	+0.39	0.01
L.M. = 0.207 IJ + 0.990	1.11	+0.40	0.01
M.N. = 0.403 IJ + 1.026	1.42	+0.58	0.001

Correlation of coefficients (r), regression equations, and the standard deviation of the scatter about the regression (SD), for the size of the G.H., H.I., J.K., K.L., L.M., and M.N. segments on the size of the I.J. segment. In some instances these calculations were made for the size of the G.H. segment on the size of the H.I. segment. These results were obtained on a group of normal men and women for the average size of these segments in 10 successive cardiac cycles and for the size of the segments in a representative inspiratory and expiratory complex.

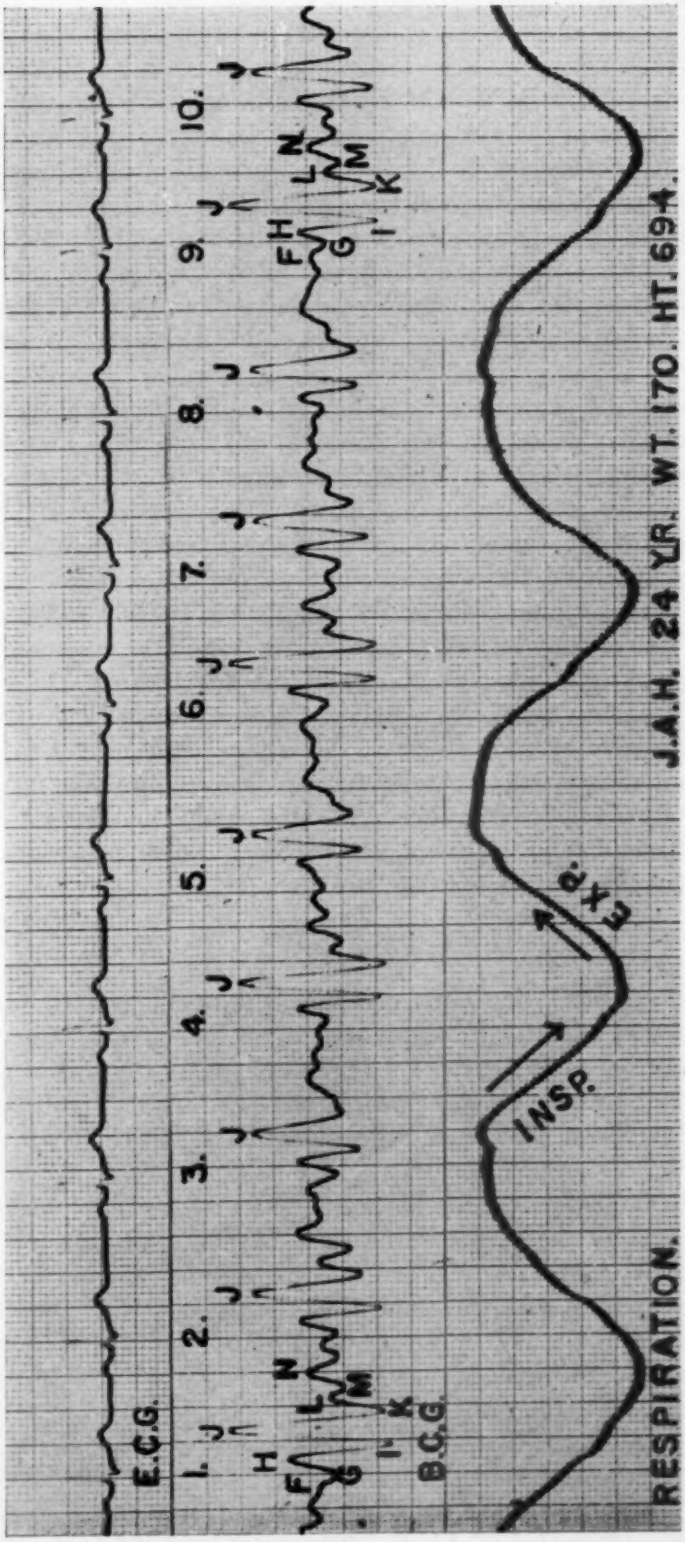


Fig. 1.—An ECG (above), a BCG (middle), and a tracing of respiratory movements (below) are shown for a normal 24-year-old man. The size of the G.H., H.I., I.J., J.K., K.L., L.M., and M.N. segments are measured in 10 successive cardiac cycles (1 to 10) and averaged. The size of these segments is also noted for a representative large inspiratory complex (No. 6) and an expiratory complex (No. 7). The method for the analysis of the form of the BCG from these measurements is described in the text. E.C.G., Electrocardiogram; B.C.G., Ballistocardiogram; Insp., Inspiration; Exp., Expiration.

The vertical heights of the G.H., H.I., I.J., J.K., K.L., L.M., and M.N. segments of the BCG were measured in millimeters in 10 successive cardiac cycles, and averaged (Fig. 1). Appropriate corrections were made to those measurements in which the calibration of the BCG deviated from the conventional standard, where a weight of 280 Gm. applied to the ballistocardiograph produces a displacement of 10 millimeters in the BCG.[1] Correlation coefficients and regression equations were calculated for the G.H., H.I., J.K., K.L., L.M., and M.N. segments on the I.J. segment, and for the G.H. segment on the H.I. segment.[12] In each instance the standard deviation of the scatter about each regression equation was also calculated. Similar estimates were made for a representative single large inspiratory complex and a small expiratory complex (Table I).

The estimation of the degree to which the size of any individual segment of a BCG differed from that predicted was made in the following manner. The vertical heights of the ballistocardiographic segments were measured in 10 successive cardiac cycles and averaged (Fig. 1). The sizes of the G.H., H.I., J.K., K.L., L.M., and M. N. segments that would be expected to occur with the size of the I.J. segment actually found were calculated from the regression equations relating the sizes of these segments to the I.J. segment in normal subjects (Table I). The difference between any segment actually found and that predicted from the regression equations was divided by the standard deviation of the regression equation (Table I). This resulted in a figure, the so-called critical ratio, which was a measure of the divergence in standard deviations of the size of the segment actually found from that predicted from the regression equation. The normal range has been arbitrarily placed at a critical ratio of plus or minus 2 (standard deviations), which is similar to the range used by Starr and associates for ballistocardiographic indices of so-called "cardiac strength."[8]

RESULTS

The coefficient of correlation between the I.J. segment and the other segments of the BCG was statistically significant in all instances and justified the establishment of normal standards for this relationship by means of regression equations (Table I). In only three instances was the level of significance for r less than $P = 0.001$. These, however, were all significant at the 5 per cent level ($P > 0.05$).

The sizes of the ballistocardiographic segments in the normal female subjects were consistently smaller than they were in the normal male subjects (Fig. 2). The relationship between the I.J. segment and the other segments of the BCG was similar in both groups, with the result that the slope of the regression equation for each segment in the female group was almost identical with that for a comparable segment in the male group (Fig. 2). It would appear from these results that the regression equations calculated from the pooling of the male and female subjects would be satisfactory for general use (Table I and Fig. 2).

The results of the correlation between the I.J. segment and the other segments of the BCG were significant for a single large inspiratory complex and a single small expiratory complex (Table I). These results are dependent upon the

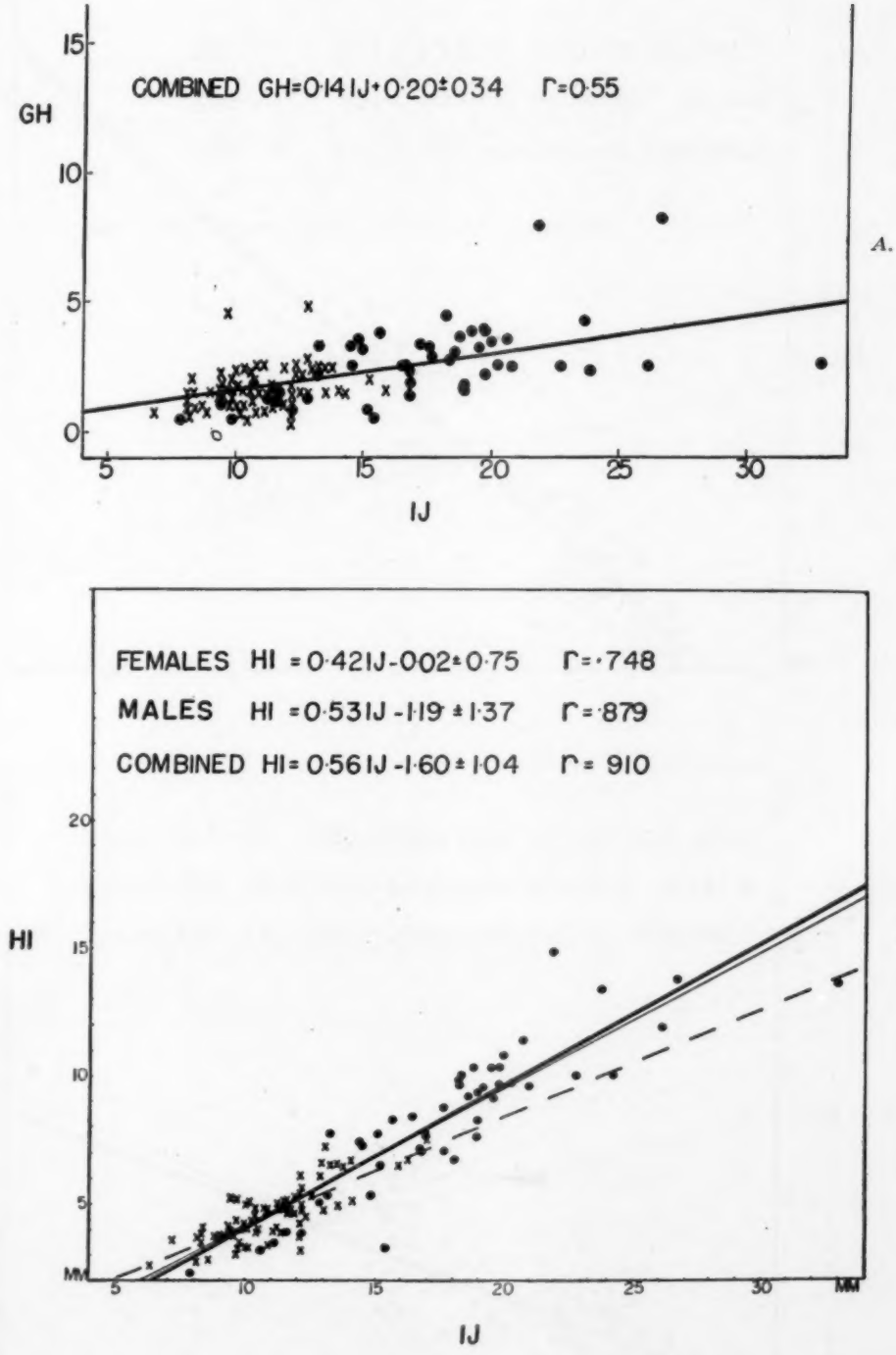
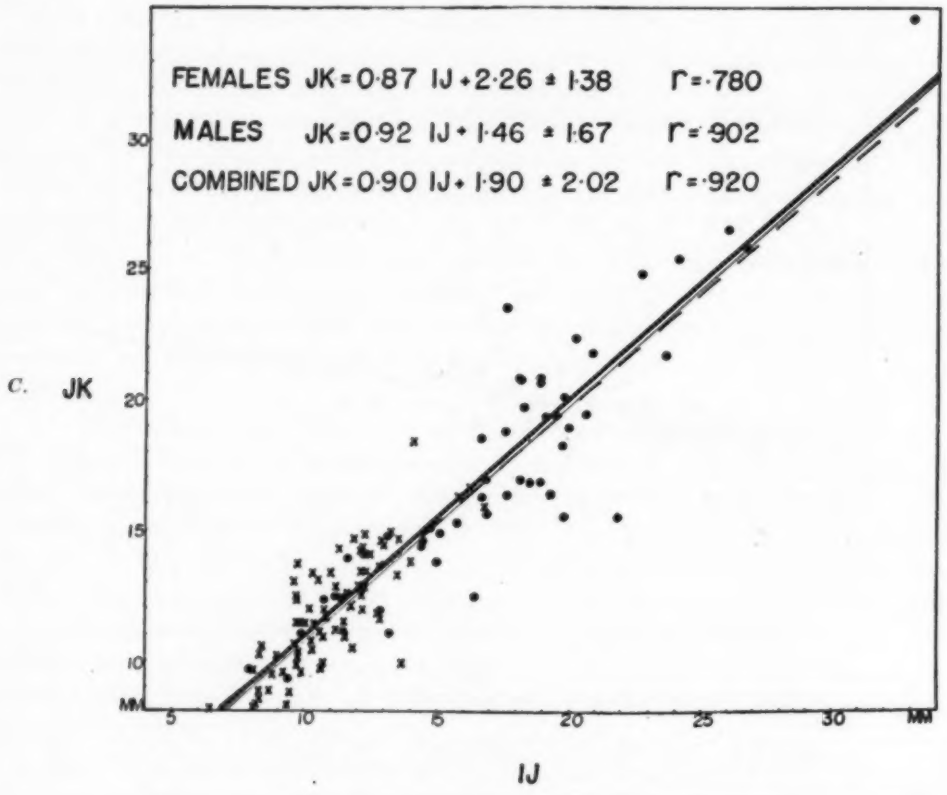


Fig. 2.—*A-F*, Regression equations have been calculated for the G.H. (*A*), H.I. (*B*), J.K. (*C*), K.L. (*D*), L.M. (*E*), and M.N. (*F*), segments on the I.J. segment where the relationship (correlation coefficient, r) between these segments was statistically significant. X = normal female subjects; 0 = normal male subjects; Broken line = Regression equation for normal female subjects; Thin continuous line = Regression equation for normal male subjects; Thick continuous line = Regression equation for male and female subjects together.



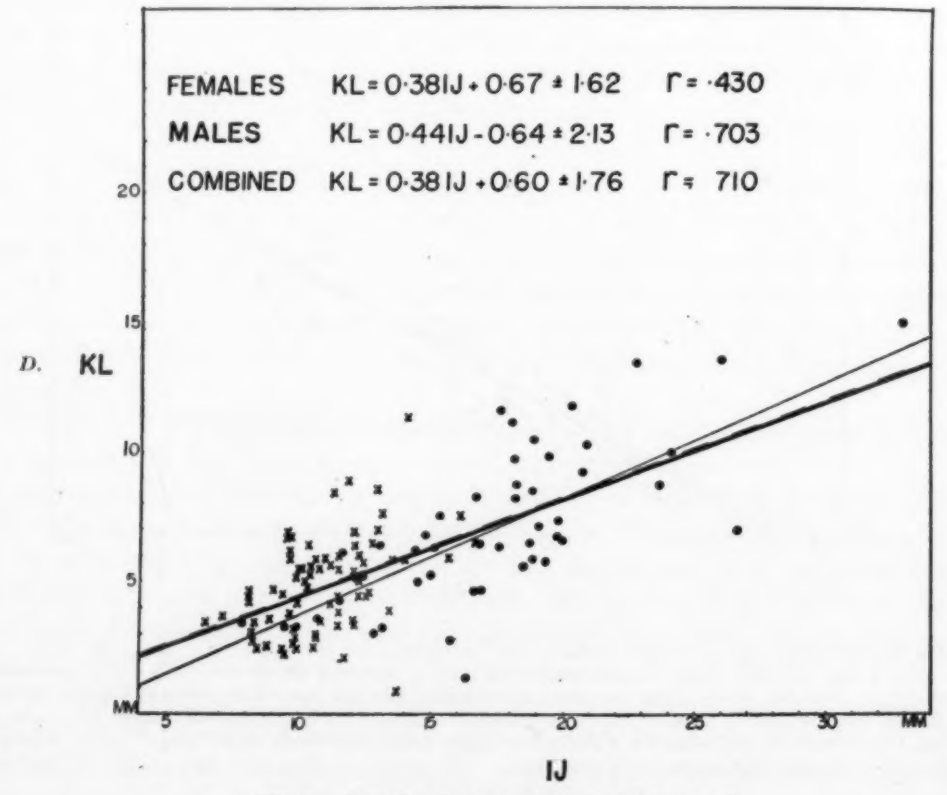
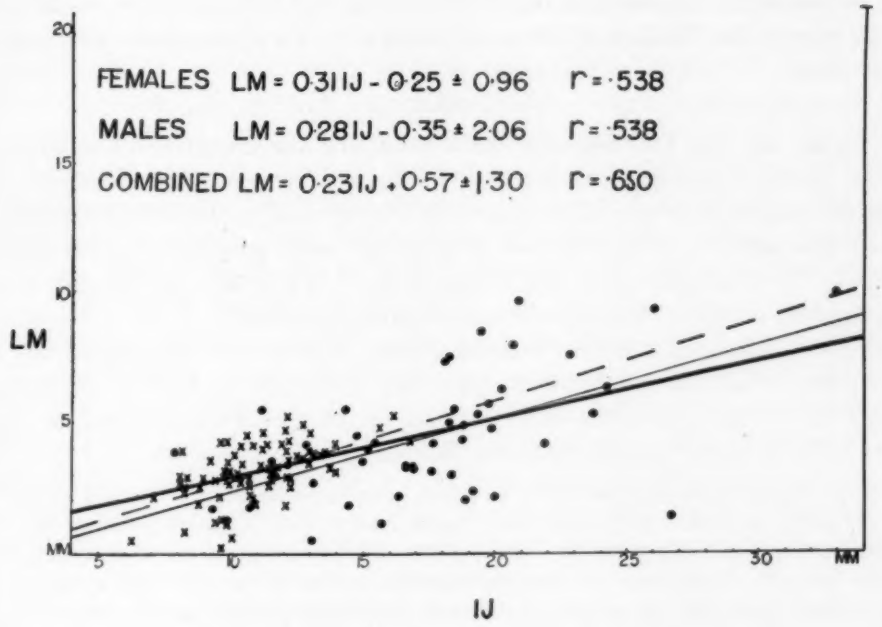


Fig. 2.—Cont'd. *(For legend see preceding page.)*



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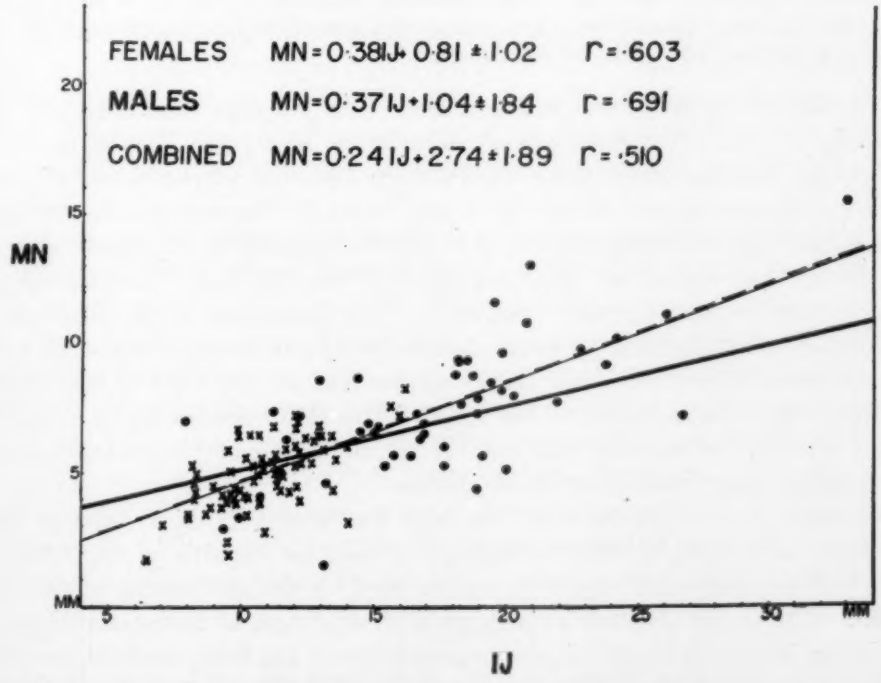

F.

Fig. 2.—Cont'd. (*For legend see page 833.*)

remarkable consistency of the form of the BCG in normal subjects. It is doubtful if such random selection of complexes would be valid in patients with heart disease, in whom the BCG may show considerable variation from one cardiac cycle to another.

DISCUSSION

The choice of the I.J. segment as a standard for comparison with other segments of the BCG in the present study was based on a number of empiric and experimental findings. The I.J. segment is a large and prominent deflection in normal records and it has been shown to correlate well with the cardiac output estimated by the Fick principle when the form of the tracing is normal[5] and to the force applied to the circulation in experimental studies.[6]

Starr and associates have summarized their reasons for the use of the I.J. segment in their study of the forces responsible for the BCG.[8] Part of this reasoning was based upon cadaver experiments, in which they simulated the function of the heart beat by forcibly expelling fluid into the circulation.

> First, to produce normal ballistocardiograms in our experiments the maximal force was delivered early in systole and these waves immediately followed; second, the general correspondence between the spread of the ballistocardiogram and the force delivered was perfectly apparent even at the experiments themselves (when the operator struck a hard blow with the mallet the ballistocardiographic spot was widely deflected, while a soft blow moved the light spot but little); third, clinical experience has shown that in healthy young adults the I and J waves are large, while in many patients with chronic heart disease, especially elderly persons with long standing coronary heart disease these waves are small or absent; fourth, the I.J., waves occur early in systole where the ballistocardiogram reflects the forces most accurately; and finally, if one employs the sum of heights or areas of two waves on opposite sides of the base line, the error of misplacing the base line is avoided or minimized.[8]

The waves of the BCG which follow the I and J waves were orginally considered to be passive after deflections.[1] Hamilton, Dow, and Remington, however, produced evidence that these waves were also due to the result of forces generated by the movement of the heart and blood.[7] The relationship between the I.J. segment and the other segments of the BCG found in this study suggests that the forces responsible for these segments of the BCG are related and proportional to each other in normal subjects. The alteration in the form of the BCG observed in patients with heart disease would be compatible with a disturbance in the relationship of the forces responsible for the form of the BCG in normal subjects. The analysis of the form of the BCG presented in this study provides a simple and empiric method for the detection of the disturbances of the forces which may result from heart disease.

The empirical interpretation of the high frequency BCG, in part at least, is inevitable. An ideal ballistocardiograph should be capable of recording accurately, without distortion, the forces generated by the movement of the heart and blood. This is not possible at present with any type of ballistocardiograph, and this is due in part to the physical characteristics of the body and the construction of ballistocardiographs. Rappaport and associates have listed the distortions that take place with a high frequency undamped ballistocardiograph, and these include amplitude distortion with respect to frequency, phase displacement, and differentiation effects.[10]

An additional confusing feature in the interpretation of the BCG is the multiplicity of forces that are generated in each heart beat. These are frequently acting at the same time with varying force and in support of or in opposition to each other. At its best, each ballistocardiographic deflection is the algebraic sum of forces acting at that time, distorted by the characteristics of the high frequency ballistocardiograph previously mentioned.

The method for the analysis of the form of the BCG that has been described is also dependent upon the conventions that have been adopted for the naming of the various deflections. It is unlikely that this method would succeed if abnormal deflections differed markedly in form and timing from those of the ballistocardiograms upon which the normal standards were based. The normal BCG is remarkably consistent in regard to the relative amplitude of the deflections and their time of occurrence. The BCG in disease may show abnormalities of the amplitudes of the deflections. However, the time of occurrence of these waves is generally very close to that which is found normally, and this is consistent with studies made by other methods of events in the cardiac cycle in normal subjects and those with heart disease. A BCG in a patient with heart disease may appear to be so distorted that it would appear to be impossible to apply the conventional lettering to the waves. Many of these tracings, however, are capable of analysis if the normal time intervals of the ballistocardiographic waves measured from the beginning of the ECG are used to establish the designation of the waves.

Deflections may, however, be abnormal in both amplitude and timing and this applies particularly to late diastolic waves observed in patients with heart disease. It has been recommended that these large diastolic waves which occur in certain abnormal records should not be given the conventional lettering, as they are believed to be due to forces quite different from those which produce the smaller diastolic waves in normal subjects.[9] The present method of analysis would be in error in such cases and should not be used where unusual deflections occur at times in the cardiac cycle where such a deflection would not normally be expected.

In the present analysis of the BCG it has been considered advisable to measure the size of the segments from trough to peak. It would be possible to make other measurements. The vertical height or depth of the various waves could have been measured from a base line. In hearts with a slow rate the base or neutral line is the horizontal portion of the tracing that is recorded late in diastole. In hearts with a faster rate it is suggested that the base line can be approximated by placing a horizontal line such that the areas of the late diastolic waves above and below it are equal.[9] From a clinical experience of abnormal records it would appear that measurements from a base line would give additional information to that which may be obtained from the analysis described in this study. Such measurements would be particularly useful in any vector study of the BCG.

SUMMARY

1. A method for the analysis of the form of the high frequency undamped ballistocardiograph has been described.

2. A sufficiently high degree of correlation exists between the I.J. segment and the other segments of the normal BCG to allow this relationship to be expressed by means of regression equations.

3. The establishment of normal standards for the relationship of the I.J. segment to the other segments of the BCG allows disturbances in the form of the abnormal BCG to be measured objectively.

SUMMARIO IN INTERLINGUA

In le ballistocardiogrammas de un serie de subjectos normal, le relation inter le dimension del segmento I.J. e le dimension del segmentos G.H., H.I., J.K., K.L., e L.M. esseva sufficientemente clar pro permitter le prediction de iste ultime deflexiones per medio del application de un equation regressional al dimension de I.J. Nos discute le rationes physiologic e empiric pro iste methodo in le analyse del forma del ballistocardiogramma. Le methodo provide un medio objective pro evalutar le forma del ballistocardiogramma in statos normal e malade.

We gratefully acknowledge help received from Drs. J. Hoffman and T. H. Bothwell.

REFERENCES

1. Starr, I., Rawson, A. J., Schroeder, H. A., and Joseph, M. R.: Studies in the Estimation of Cardiac Output in Man, and of Abnormalities in Cardiac Function, From the Heart's Recoil and the Blood's Impacts; the Ballistocardiogram, Am. J. Physiol. **127**:1, 1939.
2. Nickerson, J. L., and Curtis, H. J.: The Design of the Ballistocardiograph, Am. J. Physiol. **142**:1, 1944.
3. Dock, W., and Taubman, F.: Some Technics for Recording the Ballistocardiogram Directly From the Body, Am. J. Med. **7**:751, 1949.
4. Burger, H. C., Noordergraaf, A., and Verhagen, A. M. W.: Physical Basis of the Low-Frequency Ballistocardiograph, AM. HEART J., **46**:71, 1953.
5. Tanner, J. M.: The Construction of Normal Standards for Cardiac Output in Man, J. Clin. Invest. **28**:567, 1949.
6. Starr, I., and Schnable, T. G., Jr.: Studies Made by Simulating Systole at Necropsy. III. On the Genesis of the Systolic Waves of the Ballistocardiogram, J. Clin. Invest. **33**:10, 1954.
7. Hamilton, W. F., Dow, P., and Remington, J. W.: Relationship Between Cardiac Ejection Curves and Ballistocardiographic Forces, Am. J. Physiol. **144**:557, 1945.
8. Starr, I., Horwitz, O., Mayock, R. L., and Krumbhaar, E. B.: Standardization of the Ballistocardiogram by Simulation of the Heart's Function at Necropsy; With a Clinical Method for the Estimation of Cardiac Strength and Normal Standards for It, Circulation, **1**:1073, 1950.
9. First Report of the Committee on Ballistocardiographic Terminology, American Heart Association, Circulation, **7**:929, 1953.
10. Rappaport, M. B., Sprague, H. B., and Thompson, W. B.: Ballistocardiography. I. Physical Considerations, Circulation **7**:229, 1953.
11. Starr, I.: Essay on the Ballistocardiogram, J. A. M. A. **155**:1413, 1954.
12. Fischer, R. A.: Statistical Methods for Research Workers, ed. 7, London, 1938, Oliver & Boyd, Ltd.

THE VECTORCARDIOGRAM IN PULMONARY EMBOLISM. II

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INTRODUCTION

DURING the past several years we have had the opportunity of obtaining vectorcardiograms, in a large number of cases, with a clinical diagnosis of pulmonary embolism. This paper is concerned primarily with an evaluation of the vectorcardiographic data. Since, as is well known,[1] a major problem is the differential diagnosis between pulmonary embolism and inferior (posterior) myocardial infarction, a comparative analysis of the findings in these two conditions was also undertaken. An analysis of the electrocardiograms is included.

METHODS

The medical records of sixty-three patients with a clinical diagnosis of pulmonary embolism or pulmonary infarction in whom vectorcardiograms had been obtained were reviewed. The clinical diagnosis was acceptable in seventeen of these, and was confirmed at autopsy in six. Thirteen of the seventeen cases, including five of the six with post-mortem confirmation, were selected for this study because their vectorcardiograms presented features which we were unable to classify according to diagnostic criteria with which we were familiar.[2,3] In six cases two or more vectorcardiograms were available. Vectorcardiograms were obtained as soon as possible following the acute episode: within twenty-four hours in six cases, (4, 5, 6, 8, 9, 11), three days in one (1), six days in four (3, 7, 10, 13), nine days in one (12), and twenty-three days in one (2).

The vectorcardiographic method used in this laboratory is described elsewhere.[3]

The vectorcardiograms were projected on a viewing screen for analysis. The magnitude of the maximal rightward, leftward, anterior, posterior, superior, and inferior vectors was measured by dropping perpendiculars from the maximal points to the horizontal and the vertical axes. The following were noted: the direction of the initial, early, and terminal vectors†; the duration of the superiorly directed initial vectors, when present, expressed in seconds; the ratio of the magni-

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†Initial vectors are those at the beginning of the QRSs$\hat{E}$ loop to the right of and/or superior to the 0 point; early vectors, usually called the body of the QRSs$\hat{E}$ loop, are those to the left, inferior, anterior, and/or posterior to the 0 point; terminal vectors are those at the end of the QRSs$\hat{E}$ loop to the right of and/or superior to the 0 point.

TABLE I. SUMMARY OF THE CLINICAL AND PATHOLOGIC DATA IN THIRTEEN CASES OF PULMONARY EMBOLISM

CASE NO.	AGE SEX	CLINICAL DIAGNOSES	B.P.	CHEST X-RAY INTERPRETATION	CLINICAL COURSE	PATHOLOGIC DATA
1	52 F	Thrombophlebitis; pulmonary embolism, multiple; acute cor pulmonale	112/90	Pulmonary embolism	Sudden exitus following vena caval ligation	Adenocarcinoma of lung, multiple pulmonary emboli without infarction, multiple venous thromboses, right ventricular hypertrophy, mild
2	69 F	Diabetes mellitus, congestive heart failure, pulmonary embolism and infarction	130/60	Not done	Sudden exitus at home	
3	54 F	Ca of endometrium, pulmonary embolism and infarction	150/80	Pulmonary infarction	Pulmonary embolism following panhysterectomy	
4	68 F	Agnogenic myeloid metaplasia, thrombophlebitis, pulmonary embolism and infarction, multiple	140/90	Pulmonary infarction	Sudden exitus following discharge	Normal heart and patent coronary arteries, large pulmonary infarction, organized, old thrombosis, left iliac vein
5	26 M	Pulmonary embolism and infarction, multiple	114/72	Pulmonary embolism and infarctions	Asymptomatic on prolonged anticoagulent therapy	
6	69 F	Congestive heart failure; pulmonary embolism and infarction, multiple; paroxysmal auricular fibrillation	210/80	Pulmonary embolism and infarctions	Three episodes of pulmonary embolism	
7	49 F	Pulmonary emphysema and fibrosis, air embolism (following therapeutic pneumoperitoneum), acute cor pulmonale, coronary insufficiency	90/60	Emphysema, cor pulmonale, and pneumoperitoneum	Uneventful course following recovery from air embolism	

tude of superiorly directed initial vectors to the magnitude of the inferiorly directed vectors of the body of the loop designated as the Superior-Inferior ratio; the ratio of the magnitude of the terminal rightward vectors to the leftward vectors of the body, and the ratio of the magnitude of the terminal superior vectors to the inferior vectors of the body, designated the Rightward and Superior Terminal Appendage ratios, respectively; the direction of inscription of the QRS loops in the horizontal, sagittal, and frontal planes; angle α in the frontal plane measured as the angle subtended by the largest vector and the

TABLE I.—CONT'D

CASE. NO.	AGE SEX	CLINICAL DIAGNOSES	B.P.	CHEST X-RAY INTERPRETATION	CLINICAL COURSE	PATHOLOGIC DATA
8	44 M	Melanosarcoma of thigh, pulmonary embolism and infarction	120/80	Pulmonary embolism and infarction	Radical groin dissection followed by pulmonary embolism, expiration with pulmonary embolism 6 months later	Melanosarcoma, metastatic to all the viscera; multiple pulmonary emboli and infarctions; dilatation of the right heart
9	71 M	Arteriosclerotic heart disease, peripheral arteriosclerosis, pulmonary embolism	120/90	Negative	Femoral artery graft, followed by thigh amputation; several episodes of chest pain, tachypnea, hypotension following latter operation; sudden exitus	Extensive myocardial infarction, old, of the posterior wall and interventricular septum; nonocclusive coronary atherosclerosis; pulmonary embolism without infarction; thromboses of the abdominal aorta, iliac, and femoral arteries
10	62 F	Arteriosclerotic heart disease, acute myocardial infarction, pulmonary embolism and infarction	150/100	Pulmonary embolism and infarction	Myocardial infarction during previous admission, re-entry because of pulmonary embolism	
11	80 F	Burns of feet and buttocks, pulmonary embolism and infarction	140/90	Pulmonary infarction	Pulmonary embolism following burns, sudden exitus	
12	67 F	Pulmonary embolism and infarction, congestive heart failure, peripheral arteriosclerosis	140/90	Pulmonary infarction	Repeat episodes of pulmonary embolism, terminally cerebral and peripheral vascular occlusions	Minimal coronary atherosclerosis; subendocardial fibrosis in the interventricular septum; multiple pulmonary emboli and infarction
13	70 M	Arteriosclerotic heart disease, pulmonary embolism and infarction	90/60	Pulmonary infarction	Uneventful course	

horizontal axis; the presence or absence of unusual slowness of inscription, especially in the terminal portion of the QRSsÊ loop; the position of the S-T junction and T loops; and the direction of inscription of the T loops in each of the three planes.

Fifty cases of inferior (posterior) myocardial infarction were studied for comparison. Selection was based on an unequivocal electrocardiographic diagnosis of posterior myocardial infarction.[4] Some of these had additional evidence of infarction elsewhere or ventricular hypertrophy. The vectorcardiogram in each case was analyzed, as described above, for pulmonary embolism.

Fourteen lead electrocardiograms were done in each case in both groups, and the tracings were analyzed in detail.

RESULTS

1. *Clinical Observations in Thirteen Cases of Pulmonary Embolism.*—The data are summarized in Table I. There were nine women and four men, with an average age of sixty. Autopsies were performed on five patients.

II. *Vectorcardiographic Observations in Thirteen Cases of Pulmonary Embolism.*—The data are summarized in Table II. The initial vectors were abnormal[3] or changed following pulmonary embolism in each case. In five cases (1 to 5) initial vectors were directed superiorly, anteriorly, and to the right (Fig. 1), and in one instance (6) superiorly, anteriorly, and to the left. The

Fig. 1.—Case 1. Vectorcardiogram three days following pulmonary embolism. Note the initial superiorly directed vectors, the broad clockwisely inscribed frontal plane loop, and the large terminal appendage oriented to the right, posteriorly, and superiorly. See text and Table II. In this and the following figures H = horizontal, S = sagittal, F = frontal plane projections, and arrows indicate the direction of inscription of the QRS loop.

mean duration of the superiorly directed vectors was 0.0185 second, with a range of 0.0150 to 0.0225 second; and the mean Superior-Inferior ratio was 0.17 with a range of 0.07 to 0.32. Comparable normal mean values are 0.014 and 0.07 second, respectively.[3] In each case the duration of the superior vectors was greater than the largest values in normals for the same angle α,[3] but less compared to those in inferior myocardial infarction. The mean Superior-Inferior ratio in pulmonary embolism, likewise, was less than in inferior myocardial infarction.

The initial vectors were directed to the right, anteriorly, and inferiorly in three cases (7 to 9). This represented a change compared to a vectorcardiogram obtained prior to embolism in Case 7 (Fig. 2,*A*, *B*, and *C*). In Case 8 the initial vectors were abnormally small (Fig. 3,*A* and *B*), and in Case 9, abnormally large and associated with extensive posterior myocardial infarction.

The initial vectors were directed to the right, posteriorly, and superiorly in Case 10, and were associated with extensive anterior and posterior myocardial infarction (Fig. 4,*A*, *B*, and *C*).

In three cases (11 to 13) initial vectors were absent. The vectors at the beginning of the QRSsÊ loop were directed to the left, anteriorly, and inferiorly.

There is some abnormality of the terminal vectors in the majority of cases.[3] They were directed to the right, posteriorly, and superiorly in six cases (1, 2, 6, 7, 11, 12) (Figs. 1 and 2); the mean values of the rightward and superior vectors expressed as the Terminal Appendage ratios were 0.42 and 0.68, respectively,

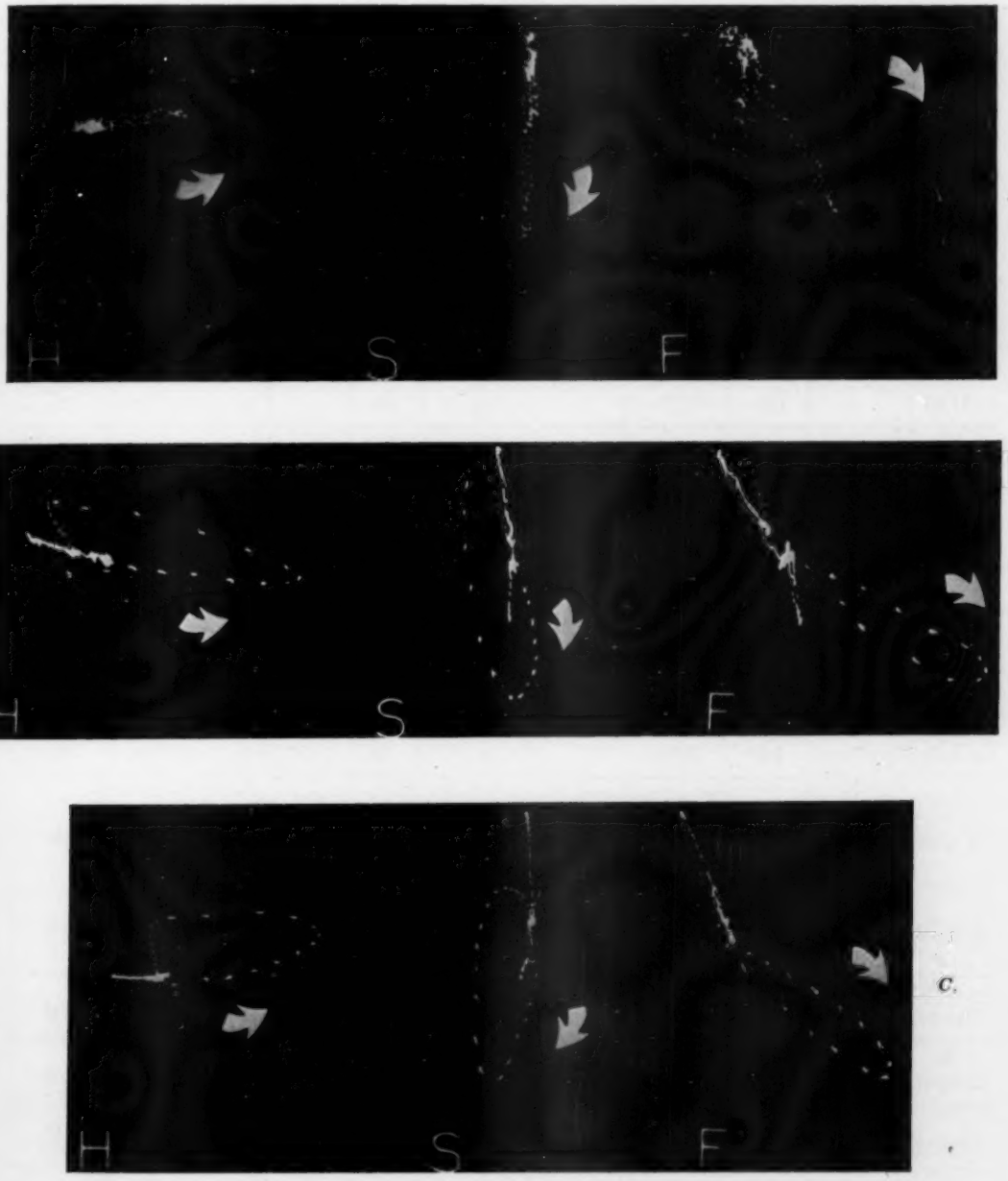

Fig. 2.—Case 7. ***A*** **shows vectorcardiogram prior to air embolism.** ***B*** **shows vectorcardiogram six days following air embolism. Note the change in direction of the initial vectors, the large terminal appendage oriented to the right, posteriorly, and superiorly, the more horizontal heart position, and the abnormal QRS-T angles.** ***C*** **shows vectorcardiogram twenty-two days following air embolism. There is a decrease in magnitude of the terminal appendage (the linear trace superior to the oval portion of the QRS loop in the frontal plane is principally T loop, compared to *B*) and a return toward a more vertical heart position. See text and Table II.**

TABLE II. SUMMARY OF THE VECTORCARDIOGRAPHIC

CASE NO. DATE OF VCG	POSITION OF VECTORS OF THE QRSS$\hat{E}$ LOOP			Su/I RATIO	DURATION OF INITIAL Su VECTORS (SECONDS)	TERMINAL APPENDAGE RATIOS		DIRECTION OF INSCRIPTION OF QRS LOOPS			ANGLE α (FRONTAL PLANE) DEGREES
	INITIAL	EARLY	TERMINAL			R	Su	H	S	F	
1. 10/17/52	RASu	$L_P^A I$	RPSu	.07	.0150	.70	1.08	cc	c	c	46
2. 9/ 8/52	RASu	$L_P^A I$	RPSu	.16	.0150	.23	.67	cc	c	c	18
3. 10/10/52	RASu	LAI	RPI	.16	.0200	.11		cc	c	c	33
4. 11/17/53	RASu	LPI	RPI	.32	.0200	.35		cc	c	c	18
5. 12/31/53		$L_P^A I$	RP_I^{Su}			.04		cc	c	cc	45
1/ 6/54		$L_P^A I$	RP_I^{Su}			.12		cc	c	cc	35
1/20/54*	RASu	$L_P^A I$	RPI	.08	.0200	.01		cc	c	cc	30
6. 10/18/54*	LASu	LPI	RPSu	.23	.0225	.38	.16	cc	c	c	6
10/22/54	LASu	LPI	RPSu	.17	.0225	.52	.08	cc	c	c	8
11/ 1/54		L_{PI}^{AO}	RPSu			.32	.39	cc	c	c	33
7. 3/ 5/54	RASu	LPI	RPSu	.10	.0200	.04	.05	cc	c	c	54
6/28/54*	RAI	$L_P^A I$	RPSu			.25	.59	cc	c	c	24
7/16/54	RAI	LPI	RPSu			.11	.23	cc	c	c	36
8. 1/12/54*	RAI	LPO	RPI			.03		cc c	c cc	cc	0
1/20/54	RAI	$L_P^A I$	RPI			.05		cc c	c	cc	24
9. 10/21/54	$RA^I{}_{Su}$	LPI						cc	c cc	c	36
10. 11/3/54	LPSu	LPI	RAO	.18	.0320	.32		cc	cc c	c	48

DATA IN THIRTEEN CASES OF PULMONARY EMBOLISM

ABNORMAL SLOWNESS OF INSCRIPTION	POSITION OF		QRS-T ANGLE (DEGREES) DIRECTION OF INSCRIPTION OF T LOOPS			MISCELLANY	VCG DIAGNOSES IN ADDITION TO ACUTE COR PULMONALE
	S-T JUNCTION	TsÊ LOOP	H	S	F		
No	000	RPI	235 ?	5 ?	74 ?		None
No	RASu	RA_I^{Su}	155 c	? ?	? cc	A-P forces greater than normal	Left ventricular hypertrophy
No	000	LPI	7 ?	5 ?	15 ?	Bulk of body of QRSsÊ loop anterior to 0 point	None
No	RAI	?	? ?	? ?	? ?	Terminal vectors cross in frontal plane	None
No	000	LAI	0 cc	0 c	10 cc	Very small initial vectors and narrow frontal loops in the 3 VCG's	None
No	000	LAI	0 cc	0 c	15 cc		None
No	RAO	LAI	0 cc	0 c	6 c	Terminal vectors cross in frontal plane in this VCG	None
No	RASu	$^{R}_{L}A_I^{Su}$	195 c	35 c	110 c	A-P forces greater than normal in the 3 VCG's	Left ventricular hypertrophy
No	RPSu	RA_I^{Su}	185 c	20 c	105 cc		Left ventricular hypertrophy
No	RPSu	RA_I^{Su}	185 c	15 c	15 c		Left ventricular hypertrophy
No	000	LOI	5 ?	8 ?	10 ?	A-P forces greater than normal in the 3 VCG's. This VCG prior to embolism	Left ventricular hypertrophy
No	RPSu	RPSu	180 cc	180 c	200 cc	Loop displaced anteriorly in VCG following embolism	Left ventricular hypertrophy
No	RASu	RPSu	192 cc	192 ?	205 c		Left ventricular hypertrophy
No	000	LAI	9 cc	50 c	30 cc	Terminal vectors cross in frontal plane in both VCG's	None
No	000	LAI	6 cc	5 c	35 cc		None
No	000	OAI	100 ?	11 ?	20 ?		Inferior and high posterior myocardial infarction
Yes	RASu	$^{R}_{L}A_I^{Su}$	175 cc	150 cc	180 c	Taken prior to embolism	Anterior, septal, inferior myocardial infarction

TABLE II.—

CASE NO. DATE OF VCG	POSITION OF VECTORS OF THE QRSsŝ LOOP			Su/I RATIO	DURATION OF INITIAL Su VECTORS (SECONDS)	TERMINAL APPENDAGE RATIOS		DIRECTION OF INSCRIPTION OF QRS LOOPS			ANGLE α (FRONTAL PLANE) DEGREES
	INITIAL	EARLY	TERMINAL			R	Su	H	S	F	
12/7/54*	RPSu	LPI	RAI	.64	.0320	.43		cc	cc	c	36
12/14/54	RPSu	LPI	RAI	.22	.0320	.56		cc	cc	c	66
11. 3/23/54		LP̂I	RPSu			1.17	2.12	cc	c	c	18
12. 9/10/54		LP̂I	ROSu			.20	.32	c	cc	c	6
13. 12/13/54*		LAI	$\overset{R}{L}$PSu			.05	1.80	cc	cc	cc	5
12/27/54		LAI	$\overset{R}{L}$PSu			.01	.87	cc	c	cc	20

VCG = vectorcardiogram; R = right, L = left, A = anterior (in reference to the patient); P = posterior, Su = superior, I = inferior; H = horizontal, S = sagittal, F = frontal; cc = counterclockwise, c = clockwise.

*VCG in closest temporal proximity to embolization in cases where several vectorcardiograms were done.

compared to mean values of 0.27 and 0.20 in the normal[3] and 0.07 and 0.11 in inferior myocardial infarction. In Case 7, these ratios changed from 0.04 and 0.05, respectively, to 0.25 and 0.59, respectively, following embolism; and with recovery they decreased to 0.11 and 0.23, respectively (Fig. 2).

The terminal vectors were directed to the right, posteriorly, and inferiorly in five cases (3 to 5, 8, 13). In three of these (4, 5, 8) the terminal vectors in the frontal plane crossed on themselves before returning to the 0 point, a feature not observed in a series of 100 normal individuals studied in this laboratory[3] (Fig. 3).

The Rightward Terminal Appendage ratio in Case 10 increased from 0.32 to 0.43 following embolism (Fig. 4).

Unusual slowness of inscription of the terminal forces occurred in only one case (10). Extensive myocardial infarction was present in this case (Fig. 4).

The QRS loops in all cases suggested unusual clockwise rotation of the heart.[5] This evidence in five cases (5, 6, 8, 10, 13) was furnished by serial vectorcardiograms (Figs. 2 and 4). Direction of inscription in the frontal plane was clockwise in ten cases (1 to 4, 6, 7, 9 to 12), and counterclockwise in three (5, 8, 13). In two of the latter (5, 8) the frontal plane loop was narrow (Fig. 3). The direction of inscription in the sagittal plane was entirely or partially counterclockwise in five cases (8 to 10, 12, 13) (Figs. 3 and 4). In Case 9 early clockwise was followed

CONT'D

ABNORMAL SLOWNESS OF INSCRIPTION	POSITION OF		QRS-T ANGLE (DEGREES) DIRECTION OF INSCRIPTION OF T LOOPS			MISCELLANY	VCG DIAGNOSES IN ADDITION TO ACUTE COR PULMONALE
	S-T JUNCTION	TsÊ LOOP	H	S	F		
Yes	RAI	$\overset{P}{L}AI$	130 cc	140 c	180 c	Loop more posterior	Anterior, septal, inferior myocardial infarction
Yes	RAI	$RA\overset{Su}{I}$	150 cc	165 c	145 c	Body of sagittal loop narrower in A-P axis	Anterior, septal, inferior myocardial infarction
No	000	LPO	50 cc	70 c	22 c		None
No	RASu	RASu	180 ?	200 ?	180 ?		Left bundle branch block
No	RPI	RAI	180 cc	15 cc	180 cc	A-P forces greater than normal	Left bundle branch block, left ventricular hypertrophy
No	RAI	RAI	160 cc	4 c	110 cc		Left bundle branch block, left ventricular hypertrophy

by counterclockwise inscription of the sagittal loop. Although Case 9 was complicated by posterior myocardial infarction, a similar inscription was not observed in the remaining forty-nine cases of inferior (posterior) myocardial infarction reviewed for comparison. On the other hand, early counterclockwise followed by clockwise inscription of the sagittal loop frequently occurs in inferior myocardial infarction, as observed in Case 10. However, following pulmonary embolism the loop in the sagittal plane in the latter case became entirely counterclockwisely inscribed (Fig. 4).

The loops in the frontal plane tended to have a horizontal position. A change toward a more horizontal position in relation to an episode of embolism was shown in the cases in which more than one vectorcardiogram was recorded (5 to 8, 10, 13)* (Figs. 2 to 4). The mean value for angle α was 21 degrees with a range of 0 to 46 degrees. In the normal vectorcardiogram the mean value is 65.69 degrees with a range of 25 to 80 degrees.[3]

In four cases (2, 6, 7, 13) the greatest anteroposterior diameter of the loop exceeded that found in normal subjects.[3] These patients had left ventricular hypertrophy.

In nine cases (1, 2, 6, 7, 9 to 13) the QRS-T angle was abnormal in one or more planes.[3] The direction of inscription of the T loops was abnormal in one or more planes in four cases (2, 6, 7, 10)[3]; normal in four (5, 8, 11, 13); and not

*In computing the mean angle and stating the range for cases in which more than one vectorcardiogram was available, the tracing in closest temporal proximity to embolization was used. In Table II the latter is indicated by an asterisk.

discernable in five (1, 3, 4, 9, 12). The S-T junction was displaced in eight cases (2, 4 to 7, 10, 12, 13); to the right in eight, anteriorly in six, posteriorly in two, superiorly in four, and inferiorly in three.

III. *Electrocardiographic Observations in Thirteen Cases of Pulmonary Embolism.*—Although available electrocardiograms were analyzed in detail, only selective features are described. The summary of the findings is given in Table III.

Fig. 3.—Case 8. ***A*** **shows vectorcardiogram less than twenty-four hours following pulmonary embolism. Note counterclockwise inscription of the sagittal plane loop, the narrow counterclockwisely inscribed frontal plane loop with crossing of the terminal vectors, and the horizontal heart position.** ***B*** **shows vectorcardiogram nine days following pulmonary embolism. There is now clockwise inscription of the sagittal plane loop and a more vertical heart position. See text and Table II.**

There was evidence of clockwise rotation in nine of the thirteen cases (1 to 6, 8, 10, 11) as judged by the presence of terminal S waves in Leads I and aV_L, late R waves in aV_R, q waves in II, III, and aV_F,[6,7] and a shift in the transitional zone to the left.[8]

There were S waves in Leads I and aV_L in eight cases (1 to 6, 8, 10), in Lead I alone in Case 11, and in aV_L alone in Cases 9 and 12. S waves were absent in both leads in Cases 7 and 13.

Qr or QR deflections in aV_R were present in six cases (1, 2, 5 to 7, 11), rSr′ deflections in two (4, 8), rS deflections in three (3, 9, 10), and QS deflections in two (12, 13). Qr or QR deflections in aV_R were not observed in any of the fifty cases of inferior myocardial infarction reviewed for this study.

The transitional zone was shifted to the left in eight cases (1, 2, 4 to 6, 8, 10, 11).

q waves were present in Leads III and aV_F in six cases (1 to 5, 10). In five of these the mean duration of the q in aV_F was 0.016 second, with a range from 0.01 to 0.02 second; the mean q/R ratio in aV_F was 0.17. In the sixth case (10) in which pulmonary embolism complicated posterior myocardial infarction,

Fig. 4.—Case 10. *A* shows vectorcardiogram following acute myocardial infarction. There are abnormal initial and early vectors characteristic of inferior and anterior myocardial infarction and an abnormal direction of inscription of the sagittal plane loop. *B* shows vectorcardiogram eight weeks following myocardial infarction and six days following pulmonary embolism. Note the increase in magnitude but not in duration of the initial superiorly directed vectors, the broad counterclockwisely inscribed sagittal plane loop, and more horizontal heart position. *C* shows vectorcardiogram thirteen days following pulmonary embolism. There is now a more narrow sagittal plane loop and a return toward a vertical heart position. See text and Table II.

TABLE III. SUMMARY OF THE ELECTROCARDIOGRAPHIC

CASE NO.	S_I	S_{aV_L}	S_{II,III,aV_F}	q_{II,III,aV_F}	q/R_{aV_F}	DURATION q_{aV_F} (SECONDS)	COMPLEX IN aV_F					COMPLEX IN aV_R			
							rS	rsr′	qrs OR qRs	qr OR qR	R	qr OR QR	rSr′	rS	QS
1.	+	+	+	+	.10	.01			+			+			
2.	+	+	+	+	.20	.02			+			+			
3.	+	+		+	.25	.02				+				+	
4.	+	+	+	+	.20	.01				+			+		
5.	+	+		+	.10	.02				+		+			
6.	+	+	+				+					+			
7.			+				+					+			
8.	+	+						+					+		
9.		+						+						+	
10.	+	+		+	.75	.04				+				+	
11.	+		+				+					+			
12.		+									+				+
13.			+				+								+

+ = present, ↓ = depressed, and ↑ = elevated.

the Q in aV_F was 0.04 second; the magnitude of the Q increased, but the duration remained unchanged following pulmonary embolism.

There were rS deflections in aV_F in four cases (6, 7, 11, 13), rsr′ deflections in two (8, 9), qRs deflections in two (1, 2), qr or qR deflections in four (3 to 5, 10), and R waves in one (12).

Prominent s waves were present in Leads II, III, and aV_F in seven cases (1, 2, 4,* 6, 7, 11, 13). These deflections increased strikingly in Case 7 following air embolism (Fig. 5,*A*, *B*, and *C*). In comparison, s waves in Leads II, III, and aV_F were absent or small in the majority of cases of inferior myocardial infarction reviewed for this study.

The approximate mean axis for the group was 29 degrees, with a range of 60 to -50 degrees. Excluding Case 10 with extensive myocardial infarction and intraventricular block, the mean value for the QRS interval was 0.076 second, with a range of 0.06 to 0.10 second. There was no instance of right bundle branch block in this series. In Case 6 the S in V_1 plus the R in V_5 was 35 mm. and less than this in the remaining cases.

*s waves were present in Leads II and III only in this case.

DATA IN THIRTEEN CASES OF PULMONARY EMBOLISM

TRANSITIONAL ZONE	MEAN AXIS (QRS) (DEGREES)	QRS INTERVAL (SECONDS)	S-T SEGMENT		INVERTED T WAVES	CLOCKWISE ROTATION
			↓ I, II	↑ III, aV_F		
V_5	60	.06	+	+	III, aV_F, V_{3R} to V_6	+
V_6	5	.07	+	+	III, aV_F, V_{3R} to V_4	+
V_2	20	.08				+
V_4	0	.06			V_{3R} to V_5	+
V_4	60	.06		+	III, aV_F	+
V_5	—10	.06	+		V_4 to V_6	+
V_3	60	.07		+	III, aV_F, V_2 to V_6	
V_4	45	.08				+
V_3	0	.09				
V_4	indeterminate	.12				+
V_6	—50	.08	+	+	III, aV_F, V_{3R} to V_3	+
V_3	40	.10				
V_1	0	.10	+		V_2 to V_6	

The S-T segments were depressed in Leads I and II in five cases (1, 2, 6, 11, 13). There were slight S-T segment elevations and inversion of the T waves in Leads III and aV_F in five cases (1, 2, 5, 7, 11). There was inversion of the T waves in the right-sided precordial leads in four cases (1, 2, 4, 11), and the left-sided precordial leads in three (6, 7, 13).

Paroxysmal auricular arrhythmias occurred in three cases (6, 9, 13). Transient P-pulmonale was noted in one case (7).

IV. *Vectorcardiographic Observations in Fifty Cases of Inferior Myocardial Infarction.*—The data was divided for purposes of analysis into Group A (forty-three cases) and Group B (seven cases). The distinguishing feature was the major direction of inscription of the frontal plane QRS loop, clockwise in Group A and counterclockwise in Group B. This division was made because preliminary observation indicated that many of the features of the QRS loops of Group A were similar to those frequently seen in acute cor pulmonale.

In the majority of cases in both groups the initial forces were directed to the right, superiorly, and anteriorly (Table IV). In two cases initial forces began inferiorly and immediately reversed their direction to a superior orientation.

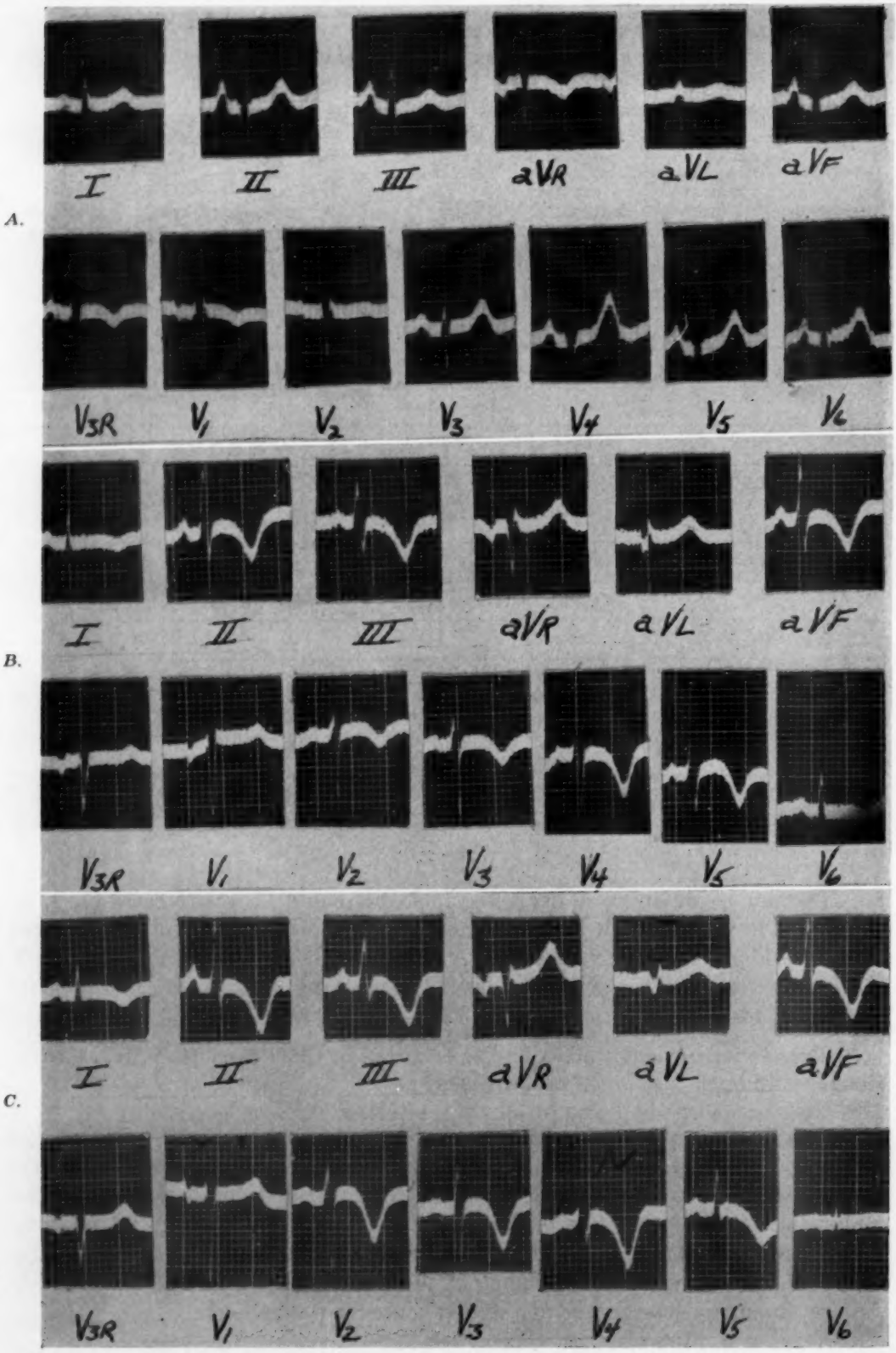

Fig. 5.—Case 7. *A* shows electrocardiogram three months prior to air embolism and is within normal limits. *B* shows electrocardiogram six days following air embolism. Note the striking increase in depth of the s waves in Leads II, III, and aV_F and inverted T waves in the limb and precordial leads. *C* shows electrocardiogram two weeks following air embolism. There is a slight decrease in depth of the s waves in Leads II, III, and aV_F and persistent inversion of the T waves. See text.

The mean duration of the initial superior vectors in Group A was 0.0338 second, and in Group B 0.0250 second. The mean Superior-Inferior ratios were 0.78 and 0.54 in Groups A and B, respectively. This contrasts with mean values of 0.014 and 0.07 second, respectively, in the normal,[3] and 0.0185 and 0.17 second, respectively, in our cases of pulmonary embolism.

TABLE IV. DIRECTIONS OF THE INITIAL AND TERMINAL VECTORS IN FIFTY CASES OF INFERIOR MYOCARDIAL INFARCTION

DIRECTION OF INITIAL VECTORS	NO. OF CASES		DIRECTION OF TERMINAL VECTORS	NO. OF CASES	
	GROUP A	GROUP B		GROUP A	GROUP B
Superior	41	7	Superior	9	4
Inferior	2	0	Inferior	34	3
Right	36	7	Right	22	2
Left	7	0	Left	21	5
Anterior	32	6	Anterior	8	0
Posterior	11	1	Posterior	35	7

Of considerable significance was the occurrence in only nine cases of Group A of superiorly directed vectors at the end of the QRS loop (Table IV). The terminal forces in the nine cases were of small magnitude with a mean superior Terminal Appendage ratio of 0.07, considerably smaller than the similar measurement in the normal[3] and in pulmonary embolism. In contrast the terminal forces in Group B attained considerable magnitude superiorly with a mean ratio of 1.20. Twenty-two cases of Group A went to the right terminally; these vectors, also, were of small magnitude with a mean Terminal Appendage ratio of 0.11 which is smaller than the corresponding measurement in the normal[3] and in pulmonary embolism.

The direction of inscription of the sagittal plane loop was clockwise in twenty-one and counterclockwise in eleven Group A cases; clockwise except for a counterclockwise initial portion in ten, and counterclockwise except for a clockwise initial portion in one. Data in the last case have been presented in Section II (above). The sagittal plane was clockwisely inscribed in six Group B cases; in the seventh case the loop was clockwisely inscribed except for an initial counterclockwise portion.

The mean value for angle α in Group A was 22 degrees with a range of 65 to -45 degrees. In Group B comparable values were 8 degrees and 84 to -90 degrees. The relationship of the duration of the initial superiorly directed vectors to angle α in the frontal plane is charted in Table V. There appeared to be no significant correlation between angle α and the duration of the initial vectors. However, compared to the normal,[3] the initial forces were markedly prolonged in every case of the present series.

TABLE V. RELATIONSHIP OF THE DURATION OF THE SUPERIORLY DIRECTED INITIAL VECTORS TO ANGLE α IN THE FRONTAL PLANE IN FIFTY CASES OF INFERIOR MYOCARDIAL INFARCTION

	Angle α (degrees)	60 to 69	50 to 59	40 to 49	30 to 39	20 to 29	10 to 19	0 to 9	0 to −9	−10 to −19	−20 to −49
GROUP A	No. of cases	6	2	2	10	4	6	3	4	4	2
	Mean duration of initial vectors (seconds)	.0317	.0293	.0293	.0270	.0241	.0243	.0284	.0382	.0484	.0272
	Angle α (degrees)	84	65	50	0	−4	−45	−90			
GROUP B	No. of cases	1	1	1	1	1	1	1			
	Duration of initial vectors (seconds)	.0348	.0248	.0270	.0248	.0248	.0293	.0135			

V. *Electrocardiographic Observations in Fifty Cases of Inferior Myocardial Infarction.*—All electrocardiograms were analyzed in detail, but only selective features are described. Thirty-nine of the forty-three Group A cases displayed qR, Qr, QR, or qrsr′ deflections in aV_F (Table VI). A terminal s wave in aV_F was noted in one case, and QS deflections in two cases.* Terminal s or S waves in aV_F were present in four cases of Group B.

TABLE VI. TYPES OF QRS COMPLEXES IN aV_F AND IN aV_R IN FIFTY CASES OF INFERIOR MYOCARDIAL INFARCTION

TYPE OF QRS COMPLEX IN aV_F	NUMBER OF CASES	
	GROUP A	GROUP B
qR or Qr or QR	33	2
qRs	0	3
qrsr′	6	0
QS	2	0
rsr′	1	1
rS	1	1

TYPE OF QRS COMPLEX IN aV_R	NUMBER OF CASES	
	GROUP A	GROUP B
rS	35	2
rSr′	6	4
QS	2	1
qr or Qr	0	0

*An rsr′ complex was present in one case.

rS deflections in aV_R were present in thirty-five cases in Group A (Table VI), rSr′ deflections in six, and QS deflections in two. There was no instance of a qr or QR deflection in aV_R. In Group B, rSr′ deflections were present in four cases. Initial r, and absent or small terminal r deflections in aV_R were the rule in the majority of cases of inferior myocardial infarction.

Twenty-three of the fifty cases had terminal s waves in Leads I and/or aV_L.

The mean duration of Q waves in aV_F in Groups A and B, respectively, was 0.0392 second, with a range of 0.02 to 0.08 second, and 0.0360 second, with a range of 0.03 to 0.04 second. The QRS interval was 0.10 second or greater in eighteen Group A cases, and 0.10 second or greater in two Group B cases.

DISCUSSION

The electrocardiogram is recognized as a useful adjunct in the diagnosis of acute cor pulmonale.[1,9-12] However, in many instances tracings are within normal limits or display nondiagnostic abnormalities.[13,14] In addition, the presence of previous abnormalities tends to obscure changes resulting from the additional insult.[15] Since positional and rotational changes play a large part in the diagnosis of acute cor pulmonale,[9,11,12] it is interesting that the vectorcardiogram is capable of revealing slight or gross changes in position and rotation despite the presence of pre-existing abnormalities.[5]

A cardinal feature in the present series is transient clockwise rotation of the QRSsÊ loop about the longitudinal axis. This change was disclosed by the vectorcardiogram in the presence of left ventricular hypertrophy, left bundle branch block, and extensive myocardial infarction. The evidence of clockwise rotation appeared in the entire QRSsÊ loop[5]; the initial vectors, representing septal and early free wall depolarization, became directed less to the right and more superiorly; rightward displacement was never marked; and in three cases the earliest vectors were directed to the left. The inscription of the body of the loop in the sagittal plane was counterclockwise in a number of instances, a feature never observed in the normal vectorcardiogram.[3] The inscription in the frontal plane was clockwise, or, with lesser degrees of rotation, narrow and counterclockwise. The terminal vectors were directed to the right with but one exception, a case with inferior myocardial infarction.

When the initial vectors were superiorly directed their magnitude and duration were greater than similar measurements in the normal[3] and less than in most cases of inferior myocardial infarction for respective angles α. These differences help to differentiate the vectorcardiograms in the three groups. In three instances the earliest forces were directed to the left, anteriorly, and inferiorly, a feature of left bundle branch block[2]; this conduction defect was thought to be present in two of these. The bodies of the QRSsÊ loops suggested clockwise rotation, i.e., there was clockwise inscription in the frontal plane and counterclockwise inscription in the sagittal plane, contrary to the usual findings in left bundle branch block.[16]

In two cases of pulmonary embolism and extensive myocardial infarction, the bodies of the loops indicated the presence of unusual clockwise rotation, as revealed by counterclockwise inscription in the sagittal plane. The transien

presence of the latter following pulmonary embolism in one of the two cases favors the view that it is an extrinsic change and not the result of myocardial infarction (Fig. 4).

Counterclockwise inscription in all but one of the horizontal plane loops furnishes an important differential point between acute cor pulmonale and right ventricular hypertrophy. In the latter, clockwise inscription in the horizontal plane and anterior displacement of the loop are the rule.[2,17]

As noted previously the terminal vectors in our cases of acute cor pulmonale were directed to the right and posteriorly in all but one case, and superiorly in six of the cases. The maximum rightward and superior vectors were of large magnitude and greater than those found in the normal[3] and inferior myocardial infarction. Their orientation and size cannot be explained by position and rotation alone, since clockwise rotation of the horizontally placed spatial loop displaces the terminal vectors inferiorly[5] rather than superiorly. In addition, although in uncomplicated left ventricular hypertrophy there is often a large appendage to the right, posteriorly, and superiorly, there is also a broad counterclockwisely inscribed frontal plane loop,[18] a feature not noted in our cases of pulmonary embolism, with one exception. Likewise, the terminal appendage in pulmonary embolism is differentiated from right bundle branch block by the absence of slow inscription and its posterior position.[19] Transient right bundle branch block was not observed in any of our cases of pulmonary embolism.

The mechanism responsible for the terminal appendage in our cases, as in normal subjects, is unknown. It has been suggested that the pulmonary outflow track, which is the thickest portion of the right ventricle, may be responsible for late rightward and superior potentials found in normal individuals and in right ventricular hypertrophy.[20,21] In this connection it is interesting that marked localized dilatation of the pulmonary outflow tract has been observed in acute cor pulmonale.[22] This suggests the possibility that local intraventricular block in this region of the right ventricle may be responsible for the terminal appendage.

Right axis deviation is a frequent finding in pulmonary embolism.[9,11,12] However, some observers have noted horizontal positions in elderly patients with pulmonary embolism and have attributed such positions to pre-existing heart disease.[15] Our observations confirm the horizontal position, but suggest that it tends to increase following pulmonary embolism. Related to these findings is the observation that the left ventricle appears as an "appendix of the right" and the heart assumes a "horizontal position" in the experimental animal following acute cor pulmonale.[22]

The abnormal angle between the long axis of the QRS and T loops noted in many of our cases corresponds to the T-wave abnormalities in the electrocardiogram of pulmonary embolism.[1,9-12,15,23] Displacement of the S-T junction to the right, anteriorly, and superiorly, observed by us, suggests ventricular ischemia.

The vectorcardiogram in inferior myocardial infarction presents several features which help distinguish it from pulmonary embolism. As noted previously, only those vectorcardiograms which display clockwise inscription in the frontal plane offer difficulty in differential diagnosis. The duration and magnitude of the initial superiorly directed vectors in myocardial infarction are

considerably greater than corresponding values found in pulmonary embolism. Furthermore, the clockwise inscription of the initial forces, as well as those of the body, are persistent, and, therefore, probably the result of intrinsic influences; i.e., in inferior myocardial infarction, unlike the situation in pulmonary embolism, there is a new balance of forces directed away from the infarcted areas. Additional differential features are the infrequent occurrence of superiorly directed terminal vectors in inferior myocardial infarction and the small magnitude of the terminal rightward vectors, which occur in half the cases. The inferior orientation of the final vectors in inferior myocardial infarction corresponds to the frequent occurrence of terminal R deflections in aV_F. It has been suggested by others that the terminal R wave represents late depolarization of living muscle overlying an infarcted area.[24,25] This is a form of local intraventricular block and, according to some observers, is more common in posterior than in other infarcts.[26]

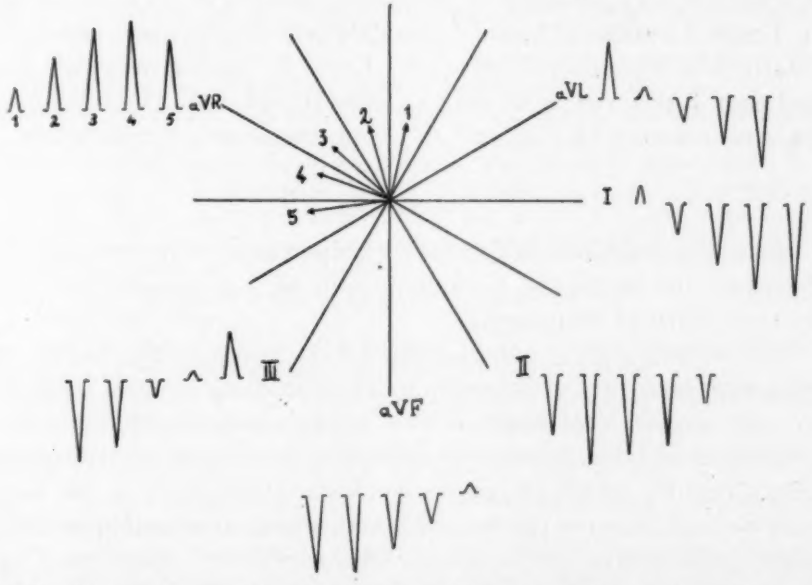

Fig. 6.—The drawing demonstrates that modest shifts of a manifest vector within a small range determine which of the limb leads will display positive or negative deflections. The scalar derivatives of the vectors projected on the various lead axes are shown adjacent to the positive ends of their respective lead axes and are arranged as shown in aV_R, numbered from *1* to *5*, corresponding to the manifest vectors 1 to 5. See text.

The electrocardiograms in nine of the thirteen cases of pulmonary embolism disclose one or more signs of clockwise rotation. Several of these signs deserve special mention. Thus, in the cases reported here, although q waves in Leads II, III, and aV_F were not abnormal in duration or magnitude, the corresponding initial vectors in the vectorcardiogram were. In the majority of cases s waves occurred in Leads I and aV_L and, as previously mentioned, were thought to be due to clockwise rotation. However, a feature of particular interest was the occurrence of s waves in Leads II, III, and aV_F, in seven cases, in two of which s waves were not present in Leads I or aV_L.

Several of these cases with s waves in Leads II, III, and aV_F displayed exceptionally large rightward, posterior, and superior appendages in the vectorcardiogram. Fig. 6 demonstrates that s waves will occur in Leads I, II, III,

aV_L, and aV_F when the manifest frontal plane vector lies in the quadrant defined approximately by the negative halves of the I and aV_F lead axis. Slight shifts of the manifest vector will determine which of the limb leads display s waves. It is not surprising that s waves in Leads II, III, and aV_F occurred in over half of our cases of pulmonary embolism, and that, in occasional instances, will appear anew in these leads in association with acute cor pulmonale. The sudden and transient appearance or increased magnitude of s waves in Leads II, III, and aV_F may have the same diagnostic significance as in Leads I and aV_L.

In the differential electrocardiographic diagnosis of pulmonary embolism and posterior myocardial infarction, the most important criteria is the rapid evolution of changes and reversion to normal in the former, versus the more persistent character of the QRS changes in the latter.[11] Other criteria which favor the diagnosis of posterior myocardial infarction include the presence of initial r waves in aV_R, absence of terminal r waves in aV_R, small or absent s waves in Leads I and aV_L, broad large Q waves in aV_F, and absence of late s waves, with few exceptions, in aV_F. Late R waves in aV_R, prominent s waves in Leads I and aV_L, q waves in Leads II, III, and aV_F, and in occasional cases, s waves in Leads II, III, and aV_F occur in acute cor pulmonale.

SUMMARY

1. Vectorcardiograms and electrocardiograms were studied in thirteen selected cases with acute cor pulmonale and fifty selected cases with inferior (posterior) myocardial infarction.

2. The following diagnostic features were noted in the vectorcardiograms of patients with acute cor pulmonale: increase in duration and magnitude of the initial vectors beyond that found in the normal, but not equalling or exceeding that occurring in inferior myocardial infarction; clockwise rotation of the QRSsÊ loop as indicated by transient counterclockwise inscription in the sagittal plane and clockwise inscription in the frontal plane; a large terminal appendage directed to the right, posteriorly, and, in some cases, superiorly; a tendency to increased horizontal electrical position; displacement of the S-T junction to the right; and abnormalities of the QRS-T angle.

3. The following features were noted in the electrocardiograms of the patients with acute cor pulmonale: clockwise rotation as judged by terminal s waves in Leads I and aV_L, late R waves in aV_R, q waves in II, III, and aV_F, and a shift in the transitional zone to the left; and s waves in II, III, and aV_F.

4. The sudden appearance following pulmonary embolism of s waves in Leads II, III, and aV_F or sudden increase in their magnitude is of diagnostic significance.

5. The vectorcardiograms of inferior myocardial infarction can be distinguished from those found in pulmonary embolism principally by the greater duration and magnitude of the initial superiorly directed vectors, and absence of superiorly oriented terminal vectors. Consequently, the corresponding distinguishing features in electrocardiograms of inferior myocardial infarction are broad large Q waves in aV_F, initial r waves in aV_R, absence or smallness of terminal r waves in aV_R, and the absence of terminal s waves in aV_F.

SUMMARIO IN INTERLINGUA

Esseva obtenite vectocardiogrammas ab 13 patientes con embolismo pulmonar. Iste vectocardiogrammas esseva analysate quantitativemente, e il esseva trovate que illos presentava aspectos distinctive de valor diagnostic. Le mesme methodo esseva applicate a vectocardiogrammas ab 50 patientes con infarcimento myocardial posterior. Le varie valores que esseva determinate in le 13 casos de embolismo pulmonar, le 50 casos de infarcimento myocardial, e 100 normal subjectos de controlo remaneva intra ben definite limites pro cata un del tres gruppos. Le resultatos signalava le signification diagnostic de aspectos electrocardiographic non previemente prendite in consideration in interpretationes differential.

REFERENCES

1. Phillips, E., and Levine, H. D.: A Critical Evaluation of Extremity and Precordial Electrocardiography in Acute Cor Pulmonale, AM. HEART J. **38**:205, 1950.
2. Wolff, L., Richman, J. L., and Soffe, A. M.: Spatial Vectorcardiography. Review and Critique, New England J. Med. **248**:810 and 851, 1953.
3. Young, E., and Wolff, L.: The Normal Vectorcardiogram. I. AM. HEART J. **51**:713,1956.
4. Myers, G. B., Klein, H. A., and Hiratzka, T.: Correlation of Electrocardiographic and Pathologic Findings in Posterior Infarction, AM. HEART J. **38**:547, 1949.
5. Wolff, L., Richman, J. L., and Soffe, A. M.: The Effect of Position and Rotation on the Cardiac Vector: An Experimental Study, AM. HEART J. **47**:161, 1954.
6. Lepeschkin, E.: Modern Electrocardiography, Vol. 1, The P-QRS-T-U Complex, Baltimore, 1950, The Williams & Wilkins Company, p. 219.
7. Goldberger, E.: Effects of Clockwise Rotation of the Heart on the Electrocardiogram, Am. J. Med. **7**:756, 1949.
8. Wilson, F. N., Rosenbaum, F. F., and Johnston, F. D.: Interpretation of the Ventricular Complex of the Electrocardiogram, Adv. Int. Med. **2**:1, 1947.
9. McGinn, S., and White, P. D.: Acute Cor Pulmonale Resulting From Pulmonary Embolism; Its Clinical Recognition, J. A. M. A. **104**:1473, 1935.
10. Murnaghan, D., McGinn, S., and White, P. D.: Pulmonary Embolism With Especial Reference to the Electrocardiogram, AM. HEART J. **25**:573, 1943.
11. Eliaser, M., Jr., and Gianseracuse, F.: The Electrocardiographic Diagnosis of Acute Cor Pulmonale, AM. HEART J. **43**:533, 1952.
12. Kuo, P. T., and Vander Veer, J. B.: Electrocardiographic Changes in Pulmonary Embolism With Special Reference to an Early and Transient Shift of the Electrical Axis of the Heart, AM. HEART J. **40**:825, 1950.
13. Love, N. S., Jr., Brugler, G. W., and Winslow, N.: Electrocardiographic Studies in Clinical and Experimental Pulmonary Embolization, Ann. Int. Med. **11**:2109, 1938.
14. Sokolow, M., Katz, L. N., and Muscovitz, A. N.: The Electrocardiogram in Pulmonary Embolism, AM. HEART J. **19**:166, 1940.
15. Dack, S., Master, A. M., Horn, H., Grishman, A., and Field, E. F.: Acute Coronary Insufficiency Due to Pulmonary Embolism, Am. J. Med. **7**:464, 1949.
16. Scherlis, L., and Grishman, A.: Spatial Vectorcardiography: Left Bundle Branch Block and Left Ventricular Hypertrophy, AM. HEART J. **41**:494, 1951.
17. Lasser, R. P., Borun, E. R., and Grishman, A.: Spatial Vectorcardiography: Right Ventricular Hypertrophy as Seen in Congenital Heart Disease, AM. HEART J. **42**:370, 1951.
18. Wolff, L.: Unpublished data.
19. Lasser, R. P., and Grishman, S.: Spatial Vectorcardiography: Right Bundle Branch Block, AM. HEART J. **42**:513, 1951.
20. Kossman, C. E., Berger, A. R., Rader, R., Brumlík, J., Briller, S. A., and Donnelly, J. H.: Intracardiac and Intravascular Potentials Resulting From Electrical Activity of the Normal Human Heart, Circulation **2**:10, 1950.
21. Kossman, C. E., Berger, A. R., Brumlík, J., and Briller, S. A.: An Analysis of Causes of Right Axis Deviation Based Partly on Endocardial Potentials of the Hypertrophied Right Ventricle, AM. HEART J. **35**:309, 1948.

22. Zuckermann, R., Rodriguez, M. I., Sodi-Pallares, D., and Bisteni, A.: Electropathology of Acute Cor Pulmonale, AM. HEART J. **40**:805, 1950.
23. Wood, P.: Pulmonary Embolism; Diagnosis by Chest Electrocardiography, Brit. Heart J. **3**:21, 1941.
24. First, S. R., Bayley, R. H., and Bedford, D. R.: Peri-infarction Block; Electrocardiographic Abnormality Occasionally Resembling Bundle Branch Block and Local Ventricular Blocks of Other Types, Circulation **2**:31, 1950.
25. Grant, R. P., and Murray, R. H.: The QRS Complex Deformity of Myocardial Infarction in the Human Subject, Am. J. Med. **17**:587, 1954.
26. Barker, J. M.: The Unipolar Electrocardiogram. A Clinical Interpretation, New York, 1952, Appleton-Century-Crofts Company, Inc., p. 333.

Erratum

In the first article of this series, entitled "The Normal Vectorcardiogram. I," by Drs. Young, Wolff, and Chatfield, which appeared in the May issue of the JOURNAL, pages 713-735, the following footnote should have appeared on page 715, with an asterisk after the heading 'Results."

***All measurements of millivolts referred to in the text and tables should be divided by 2 to obtain the actual values recorded on the oscilloscope.**

ADVANCE IN THE CLINICAL EVALUATION OF AORTIC STENOSIS BY ARTERIAL PULSE RECORDINGS OF THE NECK

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Geneva, Switzerland

THE precise diagnosis of aortic stenosis has increased in practical importance by reason of the progress made in cardiac surgery. The clinical picture, well established after years of observation and experience, has been summarized and re-evaluated in recent literature.[14] Among graphic methods used to investigate aortic valvular lesions, the arterial pulse recordings at different levels have, for a long time, claimed attention.[6,7,9,17] The carotid pulse tracing and, to a lesser degree, the humeral pulse tracing exhibit remarkable changes in aortic stenosis. Although depicted in detail in the past, these recordings have failed to become of routine use in cardiology, except by a few investigators.[5,8,12,15] The reason for this oversight may well be found in the technical aspect of the question, and in the instability of the results of such recordings.

As can be expected, each system, according to its physical characteristics, produces a different type of pulse curve. The former recorders based on pneumatic receivers and transmitters have already produced reliable tracings, but they were not easy to handle. They have been replaced gradually by electrical recorders. The use of modern intra-arterial pressure transducers which produce reliable humeral tracings cannot be extended to the carotid or subclavian arteries, because it is not advisable to place a needle in these vessels for diagnostic purposes alone. The attractive method of aortic exploration by means of a catheter, as described by Björk,[1] is not free from hazard.[2] Therefore, crystal receivers are commonly employed to collect the impluse transcutaneously. Most of the carotid tracings recently published are obtained by means of that system. Smith,[15] for instance, recently insisted on the typical systolic vibrations superimposed to the carotid sphygmographic curve in aortic stenosis. He suggested the use of an electrical setting, adapted to their elective recordings, in a limited frequency range of 40 to 70 cycles per second. Other authors[3,5] are using various types of quartz or crystal receivers and amplifiers, and obtain still different graphs which comprise a larger group of frequencies, below 40 and above 70. Unfortunately most of these crystal tracings are nonlinear in response below 30, and completely unreliable below 10. This wide range in the graphical aspect of the carotid pulse recordings is a source of confusion and controversy.

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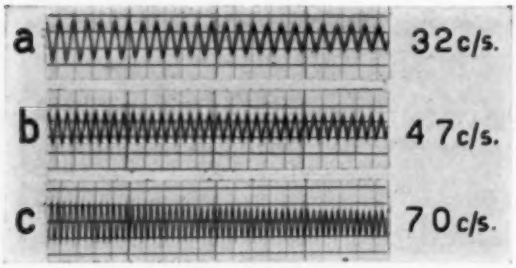


Fig. 1.—Frequency response of the oscillographic setting, composed of differential manometer, tubing, and cuff or "Marey capsule": testing by means of induced vibrations on the cuff (a-b), or "Marey capsule" (c) from a tunning fork, at 32, 47, and 70 cycles per second. The decrease in amplitude on each calibration is due to the natural extinction of vibrations in the fork. Timing: 4/100 sec.

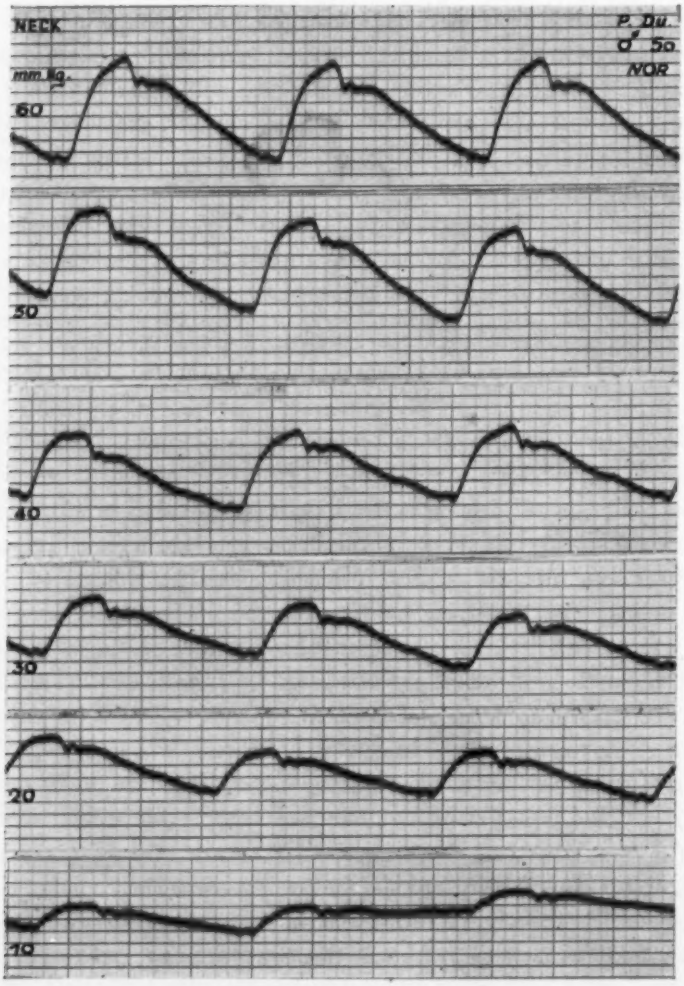


Fig. 2.—Neck oscillogram in a normal man, aged 49, at different levels of counterpressure in the cuff. Notice the great similarity of all records from 60 to 10 mm. Hg. Medium sensitivity of the system was used in this series.

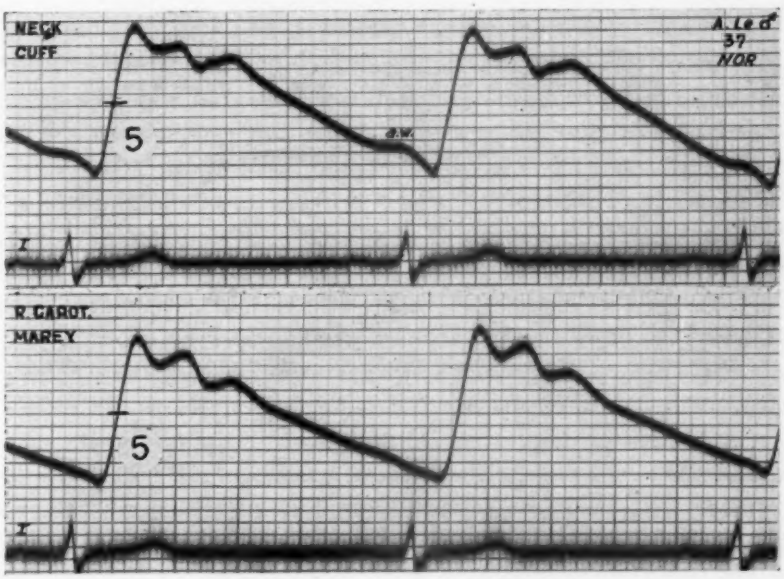


Fig. 3.—A normal man, aged 37. Comparative recordings of the neck oscillogram (above) and carotid impulse (below) in the same subject. The impulse tracing is collected by the "Marey capsule." The design of both curves is almost identical. Notice the small auricular wave (*a.w.*) on the oscillographic curve, and none on the carotid impulse tracing. The labeled number *5* indicates in 1/100 sec., the time "t" spent for the half ascent of the upstroke.

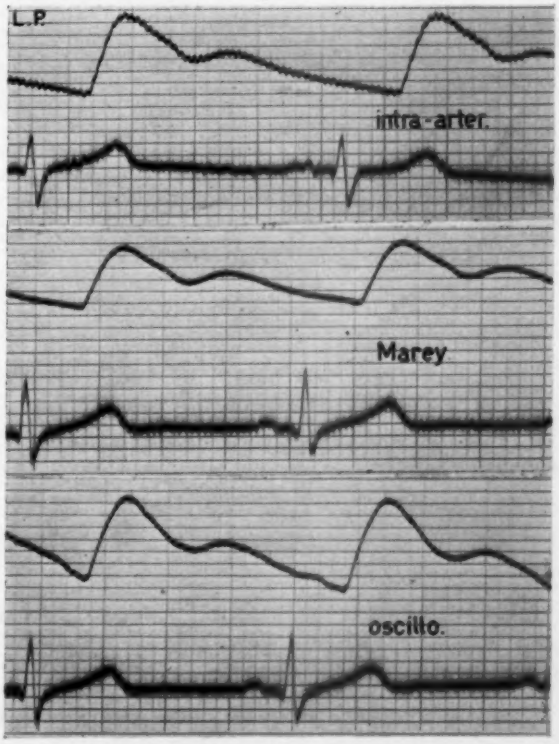


Fig. 4.—Successive recordings of the brachial artery in a normal 21-year-old man. Above: intra-arterial pressure recording (electrical). Middle: impulse recording by means of a "Marey capsule" and the differential manometer. Below: oscillometric recording, by the cuff system, connected to the same manometer. (A slight dissimilarity between the first ECG, and the two others, is attributed to inadequate grounding.)

METHOD

The device we have adopted has been recently described[10] and is a sensitive newly-built differential manometer based on O. Franck's principle. The instrument is essentially pneumatic and optical without recourse to electronics. The oscillographic unit itself has a frequency response of about 200 cycles per second, fully damped, with a sensitivity up to 2 centimeters deflection for a pressure of one centimeter water. When connected with 120 centimeter tubing to a cuff system, the frequency response goes down to a maximum of 60 cycles per second (Fig. 1). This type of apparatus will yield a linear response without phase distortion from 0 to 60 cycles per second. It has been shown in a previous paper that, at all levels of counterpressure in the cuff below diastole, the pattern of the oscillometric waves of the limbs was the same. The pulse volume only changes in accordance with the principles of Boyle's law. As already stated in our first paper, an ordinary arm cuff curled around the neck will record the pulsation of the neck vessels as well. Like those registered on the limbs, the oscillograms of the neck essentially display identical features at all infradiastolic levels avail-

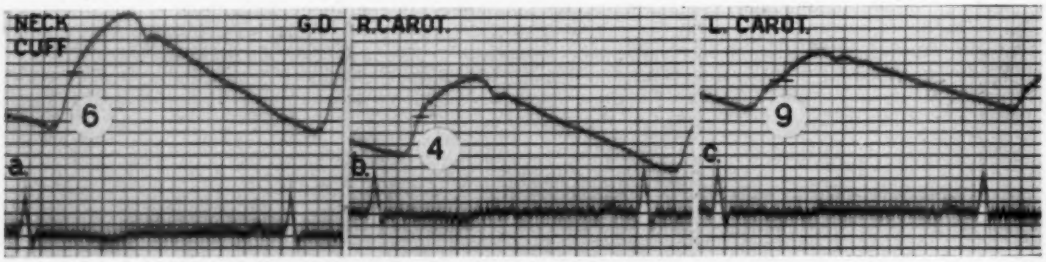


Fig. 5.—A 60-year-old man with hypertensive vascular disease. Blood pressure 230/140 and 160/110 mm. Hg after treatment. Brain circulatory disorders of left Wallenberg syndrome type. The right carotid pulsation is normal; on the left side it is distinctly diminished. The right carotid pulse tracing (*b*) shows normal contour while the left (*c*) is of a stenotic type. The neck oscillogram (*a*) represents the mean value for both.

able (Fig. 2). From 60 to 10 mm. Hg counterpressure, their amplitude while gradually decreasing in size is, nevertheless, maintained in proportion. At a counterpressure of 15 to 20 mm. Hg, which is easily tolerated by any patient, neck oscillograms of convenient amplitude are readily obtained. Their design closely resembles the carotid sphygmogram in the same subject. Two illustrations of this remarkable coincidence are given (Figs. 3 and 11). The neck oscillogram may thus be considered as an equivalent of an aterial cumulative sphygmogram at this level. It shows the classical form of the postcentral or intermediate pulse.

On the other hand, it has been shown that a record of the pulse transmitted through the arterial wall accurately reduplicates the intra-arterial pressure. Fig. 4 demonstrates this assertion for the brachial artery. This example most probably applies with the same approximation to the carotid arteries. The cuff system around the neck implies a summation of all pulsative vessels comprised in this section. If their individual sphygmographic contours are alike the resultant will have the same profile as each of them (Fig. 11). If not, it will have a mean representative value (Fig. 5). This was always borne in mind in the

present investigation, and frequent controls by separate recordings from both carotid arteries were performed. The same differential manometer was used in such controls, the cuff being replaced by a membrane receiver, a "Marey capsule."[13]

All the cases in the present study were recorded by this oscillographic device at the level of the neck, both arms, and at least one ankle. Although accurate volume standardization of the sphygmic wave has been made possible by our instrument, it was not used in the present series of patients because of the minor interest in this research. All recordings were made at a paper speed of 60 mm. per second with a simultaneous ECG to evidence rhythm abnormalities if present. Each case had a 12-lead ECG, a phonocardiogram, x-ray, and a complete clinical history.

MATERIAL

The main object of this investigation is aortic stenosis: a defect which is frequently accompanied by some degree of aortic incompetence, and occasionally by some other valvular lesion. Our series comprises twenty-eight aortic cases more or less stenotic, of which only five are considered as pure aortic stenosis.

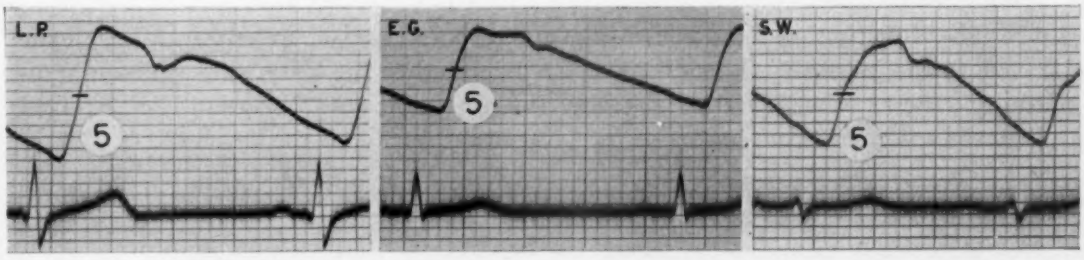


Fig. 6.—The three main types of normal neck oscillogram: (1) direct upstroke to the primary peak, (2) direct upstroke to a plateau-shaped summit, and (3) bowing upstroke after the middle ascent tending to a brief and slanted plateau. Clear-cut aortic incisura on all three tracings. Straight descending line on the second half of diastole. The auricular wave is scarcely visible. Time "t" is labeled on each curve.

The remaining twenty-three exhibit associated aortic insufficiency of different grades, eleven being more insufficient than stenotic, according to clinical standards. Five have definite signs of a mitral lesion, stenosis, or incompetence. The ages range from 16 to 75 years. One patient died and was autopsied (Case 9). The origin of aortic valvular disease is rheumatic fever in thirteen, chorea in two, and unknown in thirteen. Additional arteriosclerotic valvular damage is probably present in eleven. Two more cases are included which exemplify the discrepancies between aortic valvular pattern and other abnormal conditions. A control series of thirty normal subjects was used to formulate the normal standards of oscillographic tracings at different levels.

RESULTS

Normal Findings.—The interest of the present research is mainly concentrated on the neck tracings. Arm and ankle oscillograms were also carefully studied.

The normal carotid pulse curve reflected by the *neck oscillogram* is composed of a steep ascending limb and a variable plateau abruptly interrupted by the incisura (Fig. 6). The descending limb (catacrotic) later describes an early

smooth diastolic wave. Then it proceeds slightly uneven or straight to the foot of the next upstroke. A small auricular presystolic wave (*a.w.*, Fig. 3) is a common finding on such tracings. It follows the venous backflow from the right auricle.

Among thirty control cases three main types of normal neck oscillograms have been numbered (Fig. 6), according to variations in the ascending limb: (1) The limb goes straight to a primary peak. (2) The limb stops rather abruptly and tends to form a plateau. (3) The limb strongly reduces its vertical course (normal anacrotic inflection) and proceeds to a second or late normal summit, before the incisura. Several timing and proportion values common to the three types of tracings have been considered as normal standards. Comparing these with abnormal figures of aortic valvular diseases we attached particular attention to the primary velocity of the upstroke. The time spent from beginning of the upstroke to a point on the ascent located at half altitude of the overall curve equals four to six hundredths of a second. This delay has been named "t" value.

Above this half-way point the upstroke often continues a rapid course, with the exception of the third category of normals, where an anacrotic inflection soon supervenes.

The duration of the ejection phase can be measured from the uprise of the curve to the incisura. This duration is correlated to the heart rate. The magnitude of the rapid downstroke forming the aortic incisura is normally comprised between two and three tenths of the largest amplitude of the tracing.

The arm oscillogram is more variable from normal to normal. Its usual design is dicrotic with a first rounded, predominate summit, followed by a more or less marked second wave along the descending limb, which finally proceeds straight to the beginning of the next upstroke (Fig. 7), the "t" time for the arm oscillogram is about the same as for the neck, with variations of plus or minus one hundredth of a second.

The ankle oscillogram is of some interest also in the discussion, an example is represented in Fig. 7. It appears as a fairly sharp and symmetric summit, followed by a dicrotic wave. The time to the half-way point of the upstroke is normally comprised between seven and nine hundredths of a second.

Aortic Stenosis.—Findings in tight aortic stenosis are typically illustrated by Fig. 7, where oscillograms of the neck, arm, and ankle are matched side by side with the normal curves. The most striking deformations are seen in the *neck tracing*: (1) The upstroke is slowed, almost from the beginning, and tends to describe a straight line. (2) This limb is broken by several vibrations on its way to a delayed summit. The first of these abnormal vibrations is sometimes conspicuous, a finding which brought such tracings to be called anacrotic pulse.[11] The "t" time to half ascent, in this example, is eleven hundredths of a second; the time to the incisura is thrity-five hundredths, both figures above normal standards, especially the first. The downstroke of the aortic incisura is small-sized, and about one tenth of the total amplitude. The dicrotic wave is poorly indicated; this, in addition to the small aortic incisura, tends to make the descending limb also become a straight line.

The arm oscillogram in aortic stenosis is also frequently typical, and roughly follows the neck tracings in some of its deformations and timing disturbances. The duration for half ascent is prolonged. The dicrotic wave is erased and the incisural notch is scarcely detected. These changes lead to a flat or plateau shape of the pulse tracing, which was formerly valued as recognition of the disease.

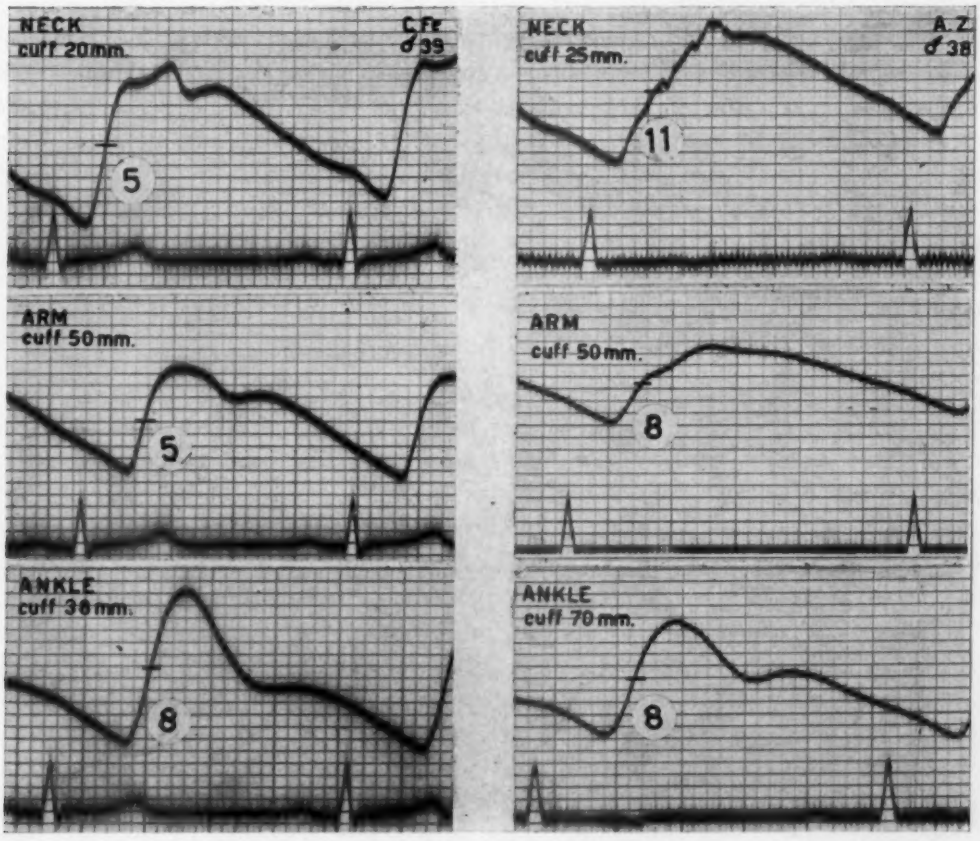


Fig. 7.—Comparative oscillograms at three levels in the normal (*A*) and in severe aortic stenosis (*B*). Great dissimilarity between the neck tracings, and close resemblance of both ankle curves. The arm curve is markedly abnormal. Typical slowing of the ascent on the neck tracing of the stenotic case. Multiple anacrotic incisuras, and straightened descending limb (Case 2, Table I).

The distal pulse represented by the *ankle oscillogram* is frequently deprived of any diagnostic characteristics. It shows normal contours, proportions, and time relations. This failure in the repercussion of the grossly abnormal conditions existing at the root of the arterial tree is commonly observed even in very stenotic cases, with a few exceptions. A necessary condition is that no important associate aortic incompetence be present. The latter defect, whenever predominate, at its turn displays other typical features. It mostly favors distortions of remote arterial pulsations. They are not considered in detail in the present paper.

The general chart of twenty-eight aortic patients graphically studied is tabulated according to the neck "t" time values. This value is labeled on each of the original records reproduced on the chart, for neck and arm oscillograms. This chart represents an attempt in classification of the degree of aortic narrowing, although pure stenosis is present in five cases only. Clinical evaluation of both stenosis and incompetence are indicated for each case, as well as the most important findings, like the grade of aortic systolic murmur and its carotid propagation, the aortic thrill, the degree of ventricular hypertrophy, eventual associated mitral lesions, heart failure, significant arrhythmia, and the blood pressure readings. The clinical evaluation 0-4 is based on the physical signs, and on the three main symptoms: dyspnea, angina pectoris, and syncope. The last example, Case 29, concerns a normal subject for comparison.

The typical oscillographic features of aortic stenosis as described above (Fig. 7) refer to Case 2 of the general chart. Similar figures are found in a number of records, like Cases 1,5,8,9,10, and 13. Less marked or other types of deformations imply a systematic discussion.

DISCUSSION

1. *The "t" Time on the Neck Tracing.*—In all twenty-eight cases of the general chart, aortic stenosis of different grades was clinically obvious twenty-four times, and suspected in four. (Cases 6, 16, 25, and 28). In the first twenty-four of the entire series, "t" exceeds the maximum normal standard of 6/100 second, the largest figure being 14/100. The last cases amount to 6/100 (Cases 25 to 27), and one to 5/100 (Case 28). This almost constant finding of a prolonged "t" time represents, therefore, a valuable measurement in the diagnosis of aotric stenosis. We presume that it also represents a quantitative approach to the degree of stenosis. The largest values clinically refer to the most marked stenosis without consideration of the presence or absence of aortic incompetence, or mitral stenosis. Cases 1 to 11, with the exception of 6, belong to this group. Another exception with reversed conditions to Case 6, may be found in Case 20, and will be discussed later. The remaining cases with a milder degree of stenosis, most often combined with aortic incompetence, and three cases with mitral defects, are also clinically related to the "t" values. In this group, however, discrepancies are more frequent and, also, of a greater diagnostic interest than in the first series. In this respect two facts must be emphasized. One is the frequent difficulty in clinical evaluation. The second is related to complementary information included in the neck oscillogram of aortic stenosis other than the "t" values. They are considered below.

From actual readings it appears that the typical slowing of the upstroke is scarcely affected by simultaneous aortic incompetence, as shown by many cases of the chart, for instance, Cases 3, 7, 11, 19, etc. The same independence is observed in the presence of associated mitral stenosis. Patient 14 is a good illustration of this fact. Here, the prominent clinical defect was mitral stenosis. Nevertheless the typical aorto-stenotic upstroke of the neck oscillogram was a surprise. It stresses the clinical difficulty in evaluation of aortic stenosis accompanying severe mitral stenosis. This problem is of great surgical importance.

Goldberg and associates[8] recently insisted on the disappointing untoward effects of commissurotomy in such conditions. Cases 11 and 24 belong to the same category, but with a lesser degree of mitral stenosis.

The consistant values of "t", for example in Cases 16 and 19, have been helpful in confirming the slight clinical suspicion of aortic stenosis. This assertion implies a relevant and proportional correlation between a prolonged "t" time, and the grade of aortic stenosis. This is our present opinion, based on several hundreds of various recordings. Of course, further investigations coupled with post mortem are necessary to confirm this. In Case 16, where the clinical diag-

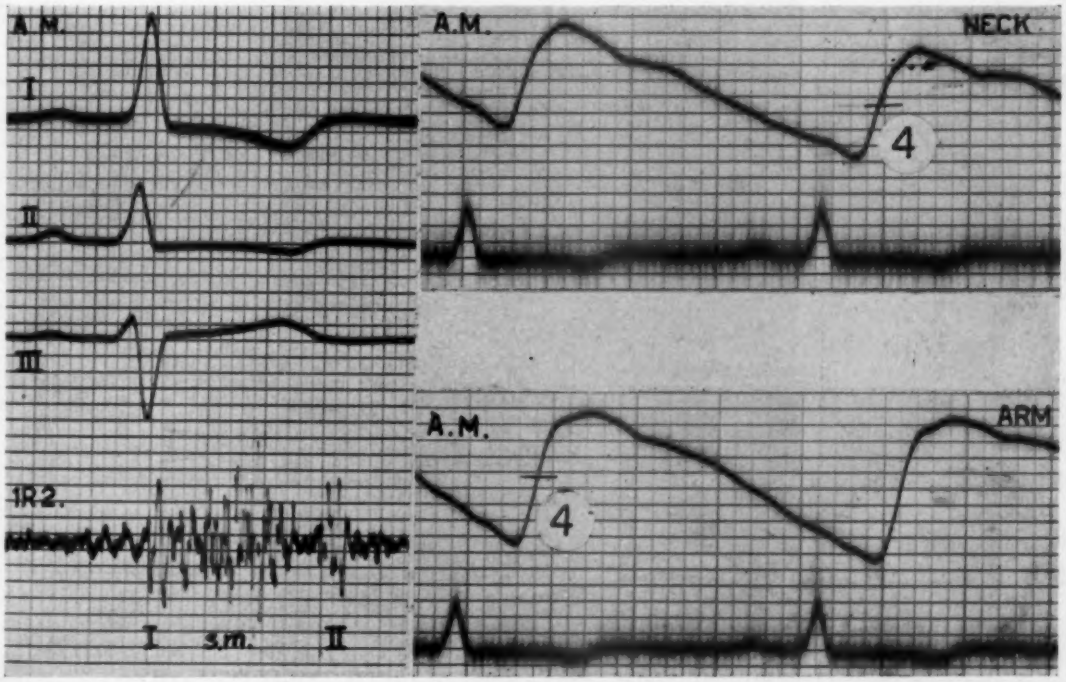


Fig. 8.—A 50-year-old man with clinically questionable aortic stenosis. Systolic murmur 5/10 at the right intercostal space. B.P. 140/90. Enlarged left ventricle. Angina pectoris. Former hypertension up to 200 mm. Hg. The diagnosis of aortic stenosis is invalidated by neck and arm oscillograms.

nosis of aortic stenosis was indefinite although a past history of rheumatic heart disease was present, we felt justified in asserting stenosis, basing our assertion on the abnormal upstroke of the neck record. Isolated hypertensive patients occasionally supplied tracings of a moderate stenotic type, without clinical support.

Of more immediate interest are those cases where aortic stenosis is suspected because of a systolic murmur at the aortic area, and where the neck recording completely fails to support the suspicion of stenosis. Such an example is illustrated on Fig. 8. It concerns a man, aged 50, complaining of angina on exertion and shortness of breath, gradually increasing in two years' time. Blood pressure is 140/90 mm. Hg. X-ray shows a big left ventricle, evidenced by the electro-

TABLE I. TWENTY-EIGHT CASES OF AORTIC STENOSIS OF DIFFERENT GRADES TABULATED ACCORDING TO THE "t" VALUES OF THE NECK OSCILLOGRAMS

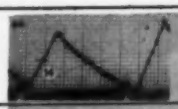
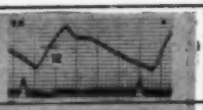
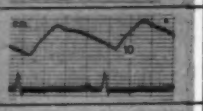
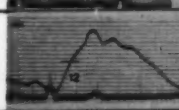
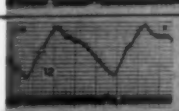
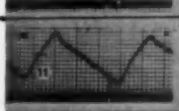
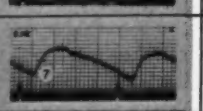
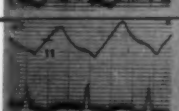
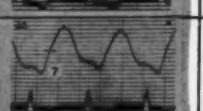
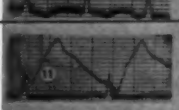
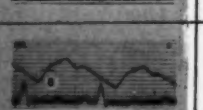
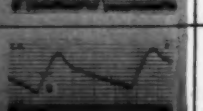
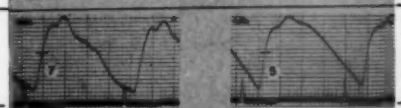
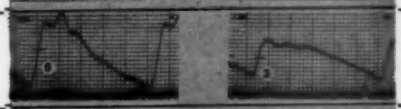
CASE N° SEX AGE	RELATIVE DEGREE OF AORTIC STENOSIS AND AORTIC INCOMPETENCE (CLINICAL) 0-4 / 0-4	NECK OSCILLOGRAM t = 1/2 ASCENT (1/100 SEC.)	ARM OSCILLOGRAM t = 1/2 ASCENT (1/100 SEC.)	AORTIC SYSTOLIC MURMUR 1-10 t = THRILL c = CAROTID PROPAGATION	LEFT VENTRICULAR HYPERTROPHY 1-4	ASSOCIATE MITRAL DEFECT 1-4 CONGESTIVE HEART FAILURE	BLOOD PRESSURE mm.Hg.
B.B. (1) m 72	4/0			6 c	2	C. H. F.	160-70
A.Z. (2) m 39	4/1			9 t c	3		105-70
C.Fl. (3) m 72	4/2			3 t c	2	C.H.F.	140-70
P.K. (4) m 51	4/1			10 t c	4		125-60 a-v. block
J.P. (5) m 46	3/1			10 c	2		150-80
G.He. (6) f 53	1-2/0			5 c	1		100-65
B.E. (7) f 57	4/2			7 c	3		120-80
J.C. (8) m 70	3/1			7 t c	3		145-85
A.F. (9) m 51	4/0			10 c	3	C. H. F.	130-90
R.C. (10) m 49	3/1			6 c	2		140-100
C.R. (11) m 46	4/2			3 t c	2	M.S. 2	115-80 L.B.B.B.
L.G. (12) f 80	2/2			4	2	C. H. F.	160-85
A.G. (13) m 58	2/1			4 c	2	C. H. F.	120-90
B.C. (14) f 51	1/2			5 c	1	M.S. 3 C. H. F.	140-90 aur. fibr.

TABLE I.—CONT'D

I.R. (15) f 55	2/2		5 c	2	M.I. 2	150·80
W.H (16) m 38	0-1/0		2	2		120·80
G.C. (17) f 59	1/0		2 c	1		140·70
G.Ha (18) m 62	2/1		6 c	2		140·90
I.S. (19) m 28	1/2		2	1		160·70
A.Rt. (20) m 54	3/1		8 c	3		l:160·80 r:145·70
G.A. (21) m 61	2/1		4 c	2		115·75
A.Rs. (22) m 35	1/3		3	2	M.I. 2	110·50
E.E. (23) m 75	1/2		2	2		150·80
J.Du. (24) m 17	2/1		5 c	2	M.S. 1	110·75
J.De. (25) m 27	0?/2		2	1		130·55
G.S. (26) f 40	1/2		4	2		120·85
B.H. (27) m 26	1/2		5	1		130·70
G.M. (28) m 22	0?/2		2	2		170·95
L.P (29) m 20	NORMAL 0/0		0	0		140·80

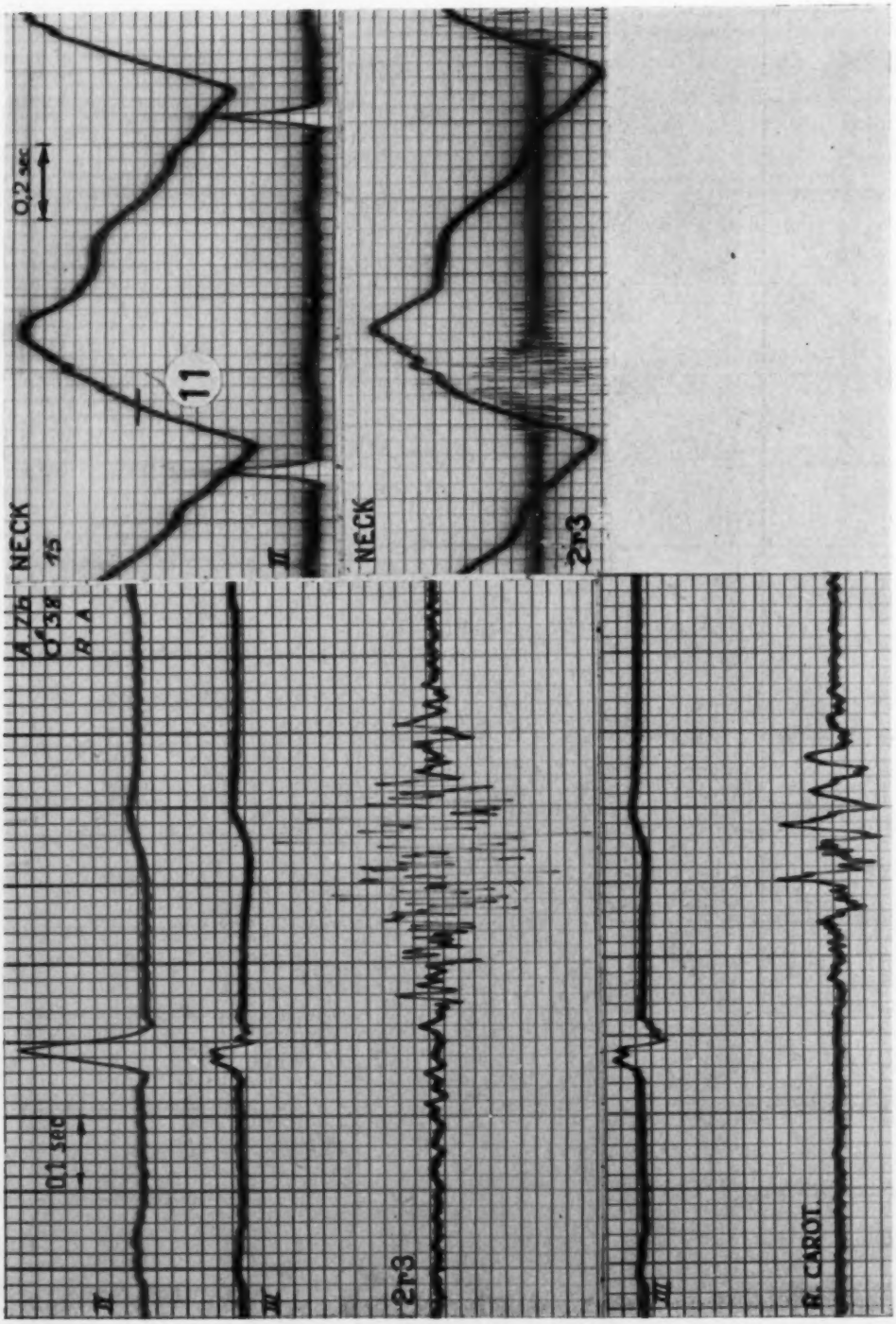


Fig. 9.—Phonocardiographic tracing at the second right intercostal space, and on the right carotid artery in a case of severe aortic stenosis. The time relations between the murmur, the neck oscillogram, and the ECG are clearly shown. The vibrations superimposed to the sphygmographic tracing are chronologically related to the slowest oscillations of the carotid phonogram. They persist beyond the acoustic part of the transmitted murmur (Case 2, Table I).

cardiogram. An aortic systolic Grade 4 murmur is present. The neck oscillogram is within normal range in regard to "t" value. This patient indeed had been hypertensive up to 200 mm. Hg for a period of time after a severe nephritis, and has been finally classified as arteriosclerotic heart disease without aortic stenosis.

As Lewis[11] has pointed out, other forms of arterial narrowing may cause the carotid or any other arterial pulse to be deformed by the type of aortic stenosis. We have seen one case with occlusive disease of both carotid arteries and a neck oscillogram of a very stenotic type, but quite normal arm oscillograms on both sides. Case 6 of the series has been a subject of discussion in the same context, because the left carotid artery is not pulsating and the clinical picture not completely consistent with the deformed neck tracing. Since both arm oscillograms also show moderate but definite patterns of a stenosis, the case has been finally classified as aortic stenosis. Some doubt still persists of an associated occlusive disease of the main stems of the aortic arch.

2. *The "t" Value of the Arm Oscillogram.*—This measurement is augmented in most highly stenotic cases. It fails, however, to follow a reliable coincidence with clinical data in the same proportion as pointed out for the neck "t" values. Many examples illustrate the discrepancy as well as the coincidence. Arm "t" is shorter than neck "t" in twenty-two cases, with two exceptions, Cases 5 and 21. It is of interest to observe that arm "t" time amounts to normal values (7/100 second or less) in seventeen cases, many of them presenting aortic stenosis of minor degree.

3. *The Anacrotic Incisura and Systolic Vibrations and Their Diagnostic Value.*—The classical description of the anacrotic pulse in aortic stenosis refers to one main incisura located on the upstroke. The latter is said to have a higher or lower location, depending upon the severity of the stenosis. We found few examples of such a predominate incisura (Cases 14 and 24), but many with multiple incisuras on the path to the delayed summit. When the amplitude of these multiple incisuras is large, the upstroke design resembles a saw (Fig. 7). In some cases the cogs are seen almost from the beginning of the upstroke (Case 9). In other cases they are mostly visible on the second half, or superior third of the ascent. They are clearly brought out in Fig. 9 (Case 2), where their time relations to the aortic and carotid phonograms are demonstrated.

In our tracings these vibrations are not a constant finding, and occur in fifteen out of twenty-eight cases. This is mainly due to the fact that their amplitude is quite variable. The recording device used in this research depicts them on film at a magnification scale identical to the scale adopted for the overall curve. This proportional response does not favor the transcription of tiny elements of the impulse. Crystal receivers or other nonlinear electric recorders often produce a more discriminative picture of these vibrations, but to the cost of the slower parts of the tracing. At any rate, the actual recording of the carotid systolic vibrations is undoubtedly of the greatest diagnostic value. Smith[15] believes that they are exclusively representative of aortic stenosis, but now we are of a different opinion since we were faced with an interesting diagnostic problem in this respect: In a man, 22 years of age, a diagnosis of aortic stenosis

was suspected on account of a harsh systolic murmur, with thrill at the second and third right intercostal spaces, and distinct carotid propagation. The neck oscillogram (Fig. 10) shows several systolic vibrations similar in design and frequency to those observed in aortic stenosis. However, the general contour of the tracing is completely different. The upstroke is rapid, time "t" only four hundredths of a second, a finding which is strictly incompatible with this lesion. The final diagnosis of interventricular septal defect has been established, and considered as much more likely in regard to the few clinical symptoms and the blowing quality of the murmur. Other considerations radiologically and electrographically complete this statement.

4. *"Delayed Summit" in Aortic Stenosis.*—The expression "delayed summit" commonly used to define the typical carotid pulse in aortic stenosis requires some comment. Here, the meaning of delayed is twofold:

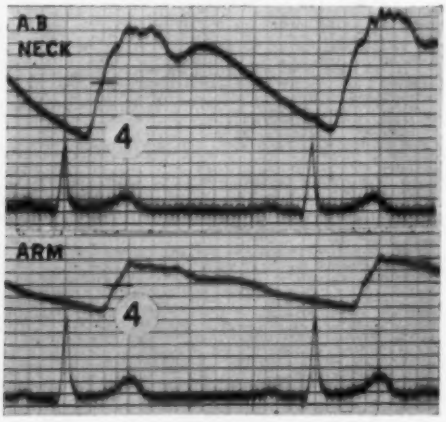


Fig. 10.—A 24-year-old man with an interventricular septal defect; asymptomatic. Grade 8/10 systolic murmur at second and third right intercostal spaces, accompanied by a thrill. Definite propagation of the murmur to the neck vessels. Carotid pulsations prominent. Blood pressure 120/80. Left ventricular hypertrophy and left axis deviation. Normal "t" value of the neck and arm oscillograms. Large systolic vibrations superimposed on the neck curve.

A. The summit is really delayed. The prolonged duration of the systole in aortic stenosis already described by other authors[16] is confirmed in the present cases. By plotting the systolic duration as a function of the rate per minute, in a series of normal cases and in the present series of stenoses, we found an average increase in systolic duration of five hundredths of a second. Since the top of the neck tracing keeps close time relations with the aortic incisural downstroke, the expression of delayed summit is etymologically correct.

B. In the majority of aortic stenotic cases there is only one summit on the neck oscillogram which corresponds to the second or late summit of many normals. On the neck tracings of the chart the top of the curve is single in all but five cases (19, 22, 25, 27, and 28). The latter cases concern patients with dominant aortic incompetence (Grades 2 to 3) and moderate stenosis (Grades 0 to 1). The more marked the stenosis, the more slanted and straight the ascent to this unique late summit. In lower grades of stenosis the upstroke is fairly steep at

first, then it bows after the first half, second third, or third quarter of the ascent to proceed directly to the summit. This broken aspect of the first limb is quite typical (Cases 15 to 17, 20, and 26). The point of bowing is correlated to the so-called normal anacrotic inflection, but it is delayed by two to four hundredths of a second. In four cases with still lesser degree of stenosis the first normal summit is maintained or just outlined (Cases 19, 22, 25, and 28). They all suffer from marked aortic regurgitation, with a wide pulse pressure.

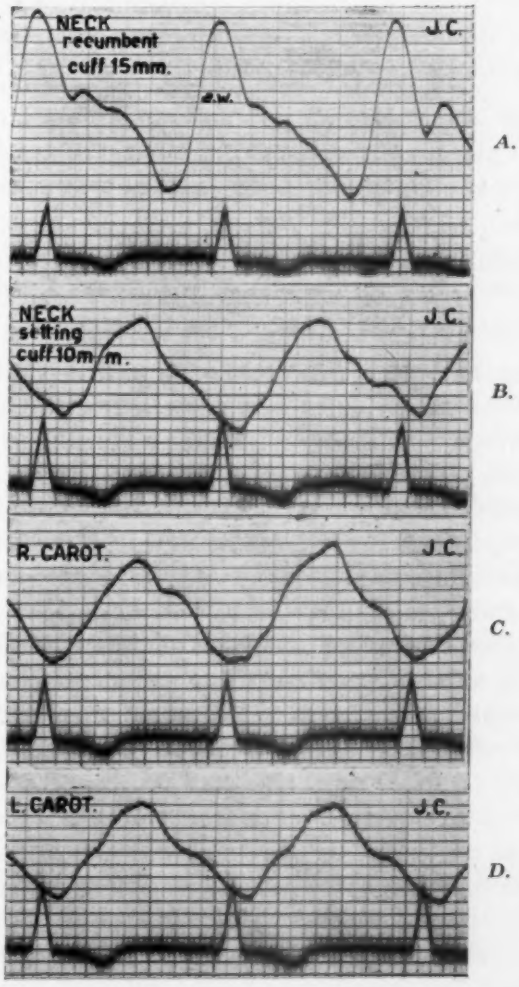


Fig. 11.—A 70-year-old man with a severe grade of aortic stenosis with mild incompetence. Congestive heart failure (Case 8, Table I). *A* and *B* are neck tracings by the cuff method with the patient recumbent and sitting. The large auricular wave (*a.w.*) disappears when the venous pressure in the jugular veins is lowered because patient is erect. *C* and *D* are individual tracings of the right and left carotid arteries recorded by the "Marey capsule." Very similar appearance of the three inferior sphygmographic tracings.

5. *The Aortic Incisura in Aortic Stenosis and Incompetence.*—As a rule the aortic incisura is of small size when the stenosis is tight (Cases 1, 2, and 5), and sometimes almost erased (Cases 8 and 12). In case of simultaneous incompetence the aortic incisural downstroke is maintained or increased (Cases 3, 4, 7, 11, and

15). It becomes conspicuous in high grades of regurgitation (Cases 19, 22, 25, and 26). There are examples where both influences, stenosis and regurgitation, interfere and probably balance one another (Cases 4, 12, and 14). In other cases the discrepancy between the features of the incisura and the clinical diagnosis are not explained (Cases 18 and 20).

There is no constant correlation between the neck and arm oscillograms in the details refering to the aortic incisura. Some are in agreement (Cases 4, 11 to 13, 20, and 27); and others are in opposition (Cases 7, 8, and 25). As already discussed by other authors[4,17] too many vascular factors are involved in this relation to discuss this problem in detail.

The techniques and recording instruments have a large importance in the appreciation of all the incisuras occurring along the tracing. Their relatively rapid frequency rate as compared to other parts of the curve gives scope to magnified deflections when crystal receivers are used. This accounts for many dissimilarities among all previously published records. This remark is relevant for the aortic incisura as well as for the anacrotic incisura and the systolic vibrations superimposed to the carotid pulse curve in aortic stenosis.

6. *The Auricular Wave of the Neck Oscillograms.*—This wave due to venous backflow at the time of auricular systole is scarcely visible on most normal oscillograms and on many pathologic tracings. Its magnitude depends upon the volume and pressure of the backflow. The altitude level of the neck in regard to the level of the heart is of great importance in its production. This assertion is self-explanatory and was proved many times in the present research work. Patients with heart failure or with increased venous pressure of any other origin may develop outstanding auricular waves when recumbent, yet these waves completely disappear when sitting. For this reason all cases suspected of failure were recorded in a more vertical position of the trunk, the change being without influence on the arterial contour itself. Some importance will probably be attached in the future to these oscillographic venous waves. The level of counterpressure existing in the cuff, 15 to 20 mm. Hg, does not prevent such waves emanating although actual pressures in the venous tract rarely amount to such magnitudes. This observation is explained by the fact that the inferior border of the cuff exerts less counterpressure on the veins with which it remains in contact. By elevating the trunk, the veins themselves become pulseless, and the pressure in the cuff as well as the marginal contact cease to play any role. This is clearly illustrated by Fig. 11.

CONCLUSIONS

1. The diagnostic importance of carotid pulse recordings in aortic stenosis is stressed especially in the present era where great strides in surgery are being made.

2. A newly built differential pneumomanometer has been applied to such recordings. Its connection with a cuff around the neck permits an easy registration of the pulsation of the vessels. The oscillogram recorded in this way at a

counterpressure of 10 to 20 mm. Hg, is most often identical to the carotid pulse recordings. It may differ in some minor details, but this does not prevent its assimilation to a carotid tracing under any circumstances.

3. Such recordings were obtained in twenty-eight cases of aortic stenosis of different grades. Five are of pure stenotic type, the others combined with aortic incompetence, and five with mitral defects. The arm and ankle oscillogram has been recorded by the same device in all cases. The neck and arm original recordings of twenty-eight patients are charted with their respective clinical data.

4. The normal neck oscillogram is a typical intermediate pulse curve with a steep ascent, one or two summits or a plateau, followed by the aortic incisura. The descending limb is slightly uneven by a protodiastolic dicrotic wave and a small presystolic auricular wave. The time spent by the upstroke to reach half of the overall altitude of the curve has been called "t" time and normally equals four to six hundredths of a second. The normal arm tracing recorded in all cases is also constituted by two summits, an early one and a dicrotic wave. Half time for the ascent of the upstroke is plus or minus one hundredth of a second the "t" value of the neck tracing in the same subject.

5. In severe aortic stenosis the neck oscillogram is typically deformed. The ascent is slower and directly reaches the top of the curve constituted by a single and delayed summit. The aortic incisura is shortened and the diastolic limb straightened up to the next rising up of the pulse. The measurement of "t" time has proved beneficial to evaluate the slow upstroke and was used in the chart for general classification. The range for "t" extends from a minimum of five to a maximum of fourteen hundredths of a second in the present series.

6. In aortic stenosis, systolic vibrations are frequently superimposed to the ascending limb of the arterial neck pulse. These vibrations are due to the turbulent streams issued from the narrowed aortic valve and transmitted to the neck. They are of great diagnostic value. Their actual finding, however, does not exclude other abnormalities like an interventricular septal defect. One example is given where the discrepancies between this type of curve and that of aortic stenosis are quite distinct.

7. Minor grades of stenosis display the same sphygmographic features, but to a lesser degree; the upstroke is not slanted as much and bows sooner or later in its ascent, to reach directly the late single summit. "t" values give a good account for the different grades of this deformation.

8. Associated aortic incompetence does not seem to influence the slowing of the upstroke due to aortic stenosis, but it definitely modifies the neck tracing at the level of the aortic incisura. The downstroke is deeper and fairly well related to the importance of the leak. Pure mitral stenosis by itself does not affect the upstroke. If mitral stenosis is associated to aortic stenosis, the slanting of the incline appears proportional to the degree of aortic stenosis. This is a relevant finding in regard to the clinical evaluation of such cases, whenever commissurotomy is anticipated.

9. The small presystolic auricular wave observed on many neck oscillograms can rise to high dimensions in case of heart failure, owing to the backflow

of a pressurized venous pulse. When the patient is placed in an upright position, recommendable for routine use, any venous pulse wave subsides.

10. In aortic stenosis the diagnostic value of the carotid tracings reflected by the neck oscillograms is confirmed. The measurement of the time spent for half ascent of the overall tracing appears as a reliable method for evaluating the degree of stenosis. In minor grades of stenosis, where this evaluation is more difficult by clinical means, the graphic judgement comes out as a still more helpful method. The differential diagnosis by means of this process is stressed. Thus several examples of clinically doubtful cases have been outlined where a clear statement in favor of, or against aortic stenosis has been graphically obtained.

SUMMARY

The principle of arterial oscillometry has been applied to the cervical vessels by connecting a cuff placed around the neck to a recording differential manometer. This instrument is essentially pneumatic without recourse to electronics. It has previously been described by the authors.

The tracings obtained at a level of counterpressure of 10 to 20 mm. Hg, which is easily tolerated, display features almost identical to the carotid sphygmograms.

This indirect process of carotid pulse recording has been applied to twenty-eight cases of aortic stenosis. The characteristics of the curves in aortic stenosis principally consist in a slowing of the upstroke with superimposed vibrations, a delayed single summit, and a straightening of the descending limb. The time spent for reaching the half-way point on the ascent has proved a ready quantitative appreciation of the slanted upstroke. It appears to correspond to the degree of stenosis. Associated valvular disease like aortic insufficiency or mitral stenosis does not modify this criterion. The value of the method in differential diagnosis is stressed.

SUMMARIO IN INTERLINGUA

Le principio de oscillometria arterial esseva applicate al vasculos cervical per connecter un manchette pneumatic, placiate circa le collo, con un manometro differential a registration. Iste instrumento es essentialmente pneumatic, sin elementos electronic. Illo ha previemente essite describite per le autores.

Le registrationes obtenite a nivellos de contrapression de inter 10 e 20 mm Hg, lo que es facilemente tolerate, exhibi tractos quasi identic con sphygmogrammas carotide.

Iste processo indirecte de registration del pulso carotide esseva applicate a vinti-octo casos de stenosis aortic. Le characteristicas del curvas in stenosis aortic consiste principalmente in un relentamento del ascendita con vibrationes superimponite, un sol summitate de occurrentia retardate, e un descendita de curso plus directe. Le tempore requirite in le ascendita usque al medietate del altitude total se ha provate significative como indice quantitative in le evalutation del ascendita e su inflexion. Illo corresponde apparentemente al grado de severitate

del stenosis. Iste criterio non es modificate per le presentia associate de morbo valvular—insufficientia aortic o stenosis mitral. Es sublineate le valor del methodo in diagnoses differential.

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REFERENCES

1. Björk, V. O., and Malmström, G.: The Diagnosis of Aortic Stenosis, AM. HEART J. **50**:303, 1955.
2. Björk, V. O., Blakemore, W. S., and Malmström, G.: Left Ventricular Pressure Measurement in Man. A New Method, AM. HEART J. **48**:197, 1954.
3. Blumberger, Kj.: Die Herzdynamik bei erworbenen Klappenfehlern, Verhandl. deutsch. Gesellsch. Kreislaufforsch., 20. Tagung, 1954, p. 43.
4. Dow, P.: The Development of the Anacrotic and Tardus Pulse of Aortic Stenosis, Am. J. Physiol. **131**:432, 1940.
5. Elsbach, H.: De clinische Diagnostiek der Aortaklepstenose, Nederl. tijdschr. geneesk. **22**:1478, 1954.
6. Feil, H. S., and Katz, L. N.: Transformation of the Central Into the Peripheral Pulse in Patients With Aortic Stenosis, AM. HEART J. **2**:12, 1926-27.
7. Feil, H. S., and Gilder, D.: The Pulse in Aortic Disease as Felt and Graphically Inscribed, AM. HEART J. **8**:4, 1921.
8. Bakst, A. A., Goldberg, H., and Bailey, C. P.: The Dynamic of Aortic Valvular Disease, AM. HEART J. **47**:527, 1954.
9. Katz, L. N., Ralli, E. P., and Cheer, S. N.: The Cardio-dynamic Changes in the Aorta and Left Ventricle Due to Stenosis of the Aorta, J. Clin. Invest. **5**:205, 1928.
10. Kettner, M. G., Ferrero, C., and Duchosal, P. W.: Clinical Investigation by the Oscillogram of Peripheral Arteries, AM. HEART J. **49**:485, 1955.
11. Lewis, Thomas: Diseases of the Heart, London, 1934, Macmillan & Co., Ltd.
12. Likoff, W., Berkowitz, D., Denton, C., and Goldberg, H.: A Clinical Evaluation of the Surgical Management of Combined Mitral and Aortic Stenosis, AM. HEART J. **49**:394, 1955.
13. Marey, E. J.: La circulation du sang a l'état physiologique et dans les maladies, Paris, 1881, G. Masson.
14. Mitchell, A. M., Sackett, Ch. H., Hunzicker, W. J., and Levine, S. A.: The Clinical Features of Aortic Stenosis, AM. HEART J. **48**:684, 1954.
15. Smith, J. E.: A Technique for Recording Carotid Artery Pulsations With Special Reference to Aortic Stenosis, AM. HEART J. **49**:428, 1955.
16. Wezler, K., and Böger, A.: Zur Mechanik der Aortenstenose. Zugleich ein Beitrag zur Analyse des Pulsus tardus, Arch. exper. Path. u. Pharmakol. **183**:387, 1936.
17. Wiggers, C. J.: Physiology in Health and Disease, ed. 5, Philadelphia, 1949, Lea & Febiger.

THE INFLUENCE OF THE CARDIAC RATE ON THE SHAPE OF THE P WAVE

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WHEREAS the descriptive morphology of the P wave is fairly complete, the nature and mechanism of action of those factors on which the shape of the auricular wave depends are still insufficiently known.

According to fundamental physiologic notions, the auricular muscular wall is stimulated by the sinus node, which is located in the right auricle. The node activates first the right and, 0.01 to 0.03 second later, the left auricle. Therefore, at least theoretically, the normal P wave should present two peaks due to the two auricular cavities which enter into action at different times. However, the time difference between the two separate electrical effects is so small that, in many instances, the peaks of the two separate auricular waves merge and form a single P wave (Pardee,[1] Scherf and Boyd[2]). Consquently, the normal P wave may present, as such, a single, peaked, or rounded, or a slightly bifid, notched form.

Different authorities give quite a variable proportion of the incidence of the various types of P waves in normal electrocardiograms. Graybiel and associates[3] found, in 1,000 healthy young aviators, the P wave in the second lead pointed in 41.1 per cent, rounded in 29.4 per cent, notched in 28.2 per cent, and diphasic in 1.6 per cent. The incidence as given by Stewart and Manning[4] for 500 healthy aviators was: rounded, 83.4 per cent, peaked, 9.8 per cent, and notched, 5.4 per cent. Attention has been given by the authorities especially to the incidence of notching. Lewis and Gilder[5] found a notched P wave in the second lead in seventeen out of fifty-two normal individuals, that is, in 32.7 per cent; Pardee saw the notched P wave in seven out of twenty-six healthy students, or 26.9 per cent in a series of 100 normal individuals; Ashman and Hull[6] have reported notched P waves in 32 per cent in a series of 100 normal individuals; Shipley and Hallaran[7] in about 30 per cent of a series of 200 healthy subjects.

So far the authorities are interested only in establishing the incidence of the different types of P waves in the normal electrocardiogram, and no attempt has been made to inquire about those factors and conditions on which the shape of the P wave may depend. In the present work a systematic study has been made of the relation existing between the shape of the P wave and the cardiac rate in normal subjects. The results of this study offer a better understanding of the morphogenesis of the differently-shaped normal P waves.

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METHOD

For a more accurate analysis of the morphologic details of the P waves, a magnifying lens was used. Only records exhibiting clear-cut P waves were made use of. Tracings inscribed with a faulty technique, or with marked muscular tremor which deform the contour of the P wave by irregular undulations were omitted. P waves were classed, as usual, into the following morphologic groups: pointed or peaked P waves; rounded P wave when the summit of P was part of a curve, or presented a horizontal course in the form of a plateau; notched P wave when the summit was divided into two clearly visible peaks, without giving special attention to the depth of the division. In a reduced number, even, of normal electrocardiograms P waves may occur that can not be enrolled in either of the groups mentioned. These P waves were classed as a special "undefined" group with a height of less than 0.5 mm. and a summit of inexact morphologic characters. Since P wave was examined in the second lead only, where it usually presents its greatest height and, thus, offers greatest convenience for a graphical analysis, we never met with shapes of the normal P wave, such as negative or diphasic ones, as frequently seen in the third lead. A total of 350 normal records were examined; they were divided into groups of fifty, according to cardiac rates ranging from 51 to 120 beats per minute.

RESULTS

Table I gives the incidence of the different types of the P waves in groups of fifty individuals, each classed according to cardiac rates.

TABLE I. PERCENTAGE OF INCIDENCE OF DIFFERENT P WAVES AT DIFFERENT CARDIAC RATES IN GROUPS OF 50 NORMAL SUBJECTS

CARDIAC RATE (MIN.)	PEAKED (%)	NOTCHED (%)	ROUNDED (%)	UNDEFINED (%)
51–60	52	32	10	6
61–70	44	20	26	10
71–80	36	14	40	10
81–90	28	12	56	4
91–100	16	16	68	0
101–110	14	12	74	0
111–120	10	4	82	4

Peaked P waves.—The greatest incidence reaching 52 per cent was found in the group of lowest cardiac rates. The incidence decreases with increasing cardiac rates and reaches its lowest value of 10 per cent in the group with the highest cardiac rate. The relative decrease of the incidence of the peaked P wave from lowest to highest cardiac rate amounted to more than five times.

Notched P Waves.—The incidence follows the same quantitative rules as that of peaked P waves. The greatest incidence was again found in the group of the lowest cardiac rate, and the lowest incidence of 4 per cent only in the group of the highest cardiac rates. The relative decrease of the incidence amounted to eight times.

Rounded P Waves.—The incidence of this variety of P wave was opposite that of the two other types: the incidence was greater the higher the cardiac rates. The relative increase of the rounded P wave from lowest to highest cardiac rate amounted to more than eight times.

Undefined P Waves.—The incidence of undefined P waves was irregular. There was no constant relation between this type of P wave and the cardiac rates, although there was a tendency to a smaller incidence with higher cardiac rates: an incidence of 9 per cent in the groups of 51 to 80 beats, and 2 per cent in the groups of 81 to 120 beats.

To summarize the results; incidence of the peaked and notched P waves is inversely proportionate to the cardiac rate, whereas incidence of the rounded P wave is directly proportioned to the cardiac rate. Since the incidence of the different shapes of P waves varies at different cardiac rates, it becomes evident that statements about the incidence of the various types of P waves in general terms, i.e., without referring to cardiac rates, are as it turns out now devoid of any value.

DISCUSSION

In the normal electrocardiogram the height of the P wave depends upon the size and position of the auricles, the functional condition of the auricular myocardium, and the contact with good or poor conducting structures of the surrounding medium. Notching is considered a normal variant. However, when present in a marked form and with a distance between the two peaks greater than 0.03 second, it represents the graphical expression of abormal intra-auricular conduction.

As generally known, mitral valvular diseases cause, by left auricular hypertrophy and dilatation, the appearance of enlarged, widened, and, in about 60 per cent of the cases, also markedly notched P waves in Leads I and II (Pardee[1]). Hypertrophy of the right auricle as in cases of cor pulmonale, causes peaked P waves of increased height in Leads II and III (Winternitz[8]). These examples chosen from clinical pathology remind us of the influence of anatomic factors on the shape of the P wave. On the other hand, functional factors of quite different origin also may alter its shape in a definite sense. Disturbances of the intra-auricular conduction due to an altered sequence of stimulation in the auricular muscular wall produce changes in the shape of the P wave, which also have been experimentally induced by ligature of the connecting fibers between the sinus node and the auricular wall (Rothberger and Scherf[9]). Autonomic nervous stimuli alter the cardiac rate, height, and shape of P and T waves (Rothberger and Winterberg[10]). Furthermore, changes in the electrolyte composition of the myocardium are usually accompanied by electrocardiographic alterations. Thus, hypocalcemia causes, among other modifications, tall, peaked P waves (Weller and associates,[11] Martin and associates,[12] and Surawicz and Lepeschkin[13]). Sodi-Pallares and Marsico,[14] when studying the electrocardiographic alterations in cases of congenital heart diseases, concluded that a low oxygen saturation of the arterial blood and a high systolic pressure in the right ventricle are those factors which produce the most striking P-wave changes. How-

ever, the influence the cardiac rate has on the shape of the P wave and its distribution at different cardiac frequencies, the object of the present study, has not been reported previously.

As shown in the present paper incidence of peaked and notched P waves obey the same rule, whereas incidence of rounded P waves follows an opposite rule. Peaked and notched P waves have their greatest incidence of 52 and 32 per cent, respectively, at the lowest cardiac rate, from 51 to 60; the combined incidence of these two varieties of P waves represents 84 per cent of all the individuals observed. On the contrary, rounded P waves have their lowest incidence of 10 per cent at the slowest cardiac rate. At the highest cardiac rate of 111 to 120 beats the distribution of the different types of P waves is just the opposite of that observed at lowest heart rate: instead of 84 and 10 at lowest heart rate, we have 14 and 82 at highest rate. So quantitative is the dependence of the incidence of different varieties of P waves from the heart rate.

The significance of the cardiac rate for the incidence of the different varieties of P waves was hitherto unknown, and a comparison with the published data of the authorities referring to this incidence is difficult, since one of its fundamental factors hitherto has been omitted. But we shall refer here to Graybiel and associates[3] who analyzed the electrocardiogram of 1,000 healthy aviators with an average cardiac rate of 64 per minute; they found rounded P waves in 29, pointed P waves in 41, and notched P waves in 28 per cent. The figures of our Table I for rounded, peaked, and notched P waves are 26, 44, and 20 per cent, respectively, at a cardiac rate of 61 to 70. This is a satisfactory agreement between the results of the two studies.

The difference in the distribution of the various P wave types according to cardiac rates must be due to peaked and notched P waves being transformed, under certain circumstances, into rounded P waves. At low cardiac rates the P wave is represented overwhelmingly by its primitive types, which are the peaked and the notched waves; the latter is the most "physiological" one (two cavities, two peaks). The two represent together 84 per cent of all cases. With increasing cardiac rates the incidence of the peaked and notched P waves decreases, and that of the rounded P waves increases almost quantitatively, at the same degree but in opposite direction. This transformation, or more pronounced combination, takes place at the highest cardiac rate from 111 to 120 beats, in 82 per cent of all the cases. Therefore it must be concluded that the form of the P wave is not constant and may vary with the cardiac rate.

SUMMARY

1. The cardiac rates exert a definite influence on the shape of the P wave. Low cardiac rate favors peaked and notched P waves, whereas high cardiac rate favors the rounded P wave.

2. Incidence of pointed and notched P waves obeys the same rule of dependence on the cardiac rate, whereas incidence of the rounded P wave obeys an opposite quantitative rule.

3. At low cardiac rates, as 51 to 60 beats per minute, the pointed and notched P waves prevail, representing together 84 per cent of all cases, whereas rounded

P waves are present in 10 per cent only. At high cardiac rates, as 111 to 120 beats per minute, the rounded P wave prevails, representing 82 per cent of all cases, whereas the peaked and notched P waves together form only 14 per cent.

4. Consequently, in addition to different anatomic and functional circumstances, the cardiac rate must be taken into consideration as one of those fundamental factors which determine the shape of the P wave.

SUMMARIO IN INTERLINGUA

1. Le frequentia cardiac exerce un definite influentia super le forma del unda P. Basse frequentias cardiac tende a associar se con undas P de forma crestate e indentate; alte frequentias cardiac es favorabile a formas ronde del unda P.

2. Le frequentia de crestate undas P e le frequentia de indentate undas P obedi al mesme regula de dependentia ab le frequentia cardiac; le frequentia del ronde undas P obedi a un contrari regula quantitative.

3. A basse frequentias cardiac—i.e. a inter 51 e 60 pulsos per minuta—il ha un prevalentia de crestate e indentate undas P. Insimul illos representa 84 pro cento del casos durante que ronde undas P se trova in solmente 10 pro cento del casos. A alte frequentias cardiac—i.e. a inter 111 e 120 pulsos per minuta—il ha un prevalentia de ronde undas P. Illos representa 82 pro cento del casos durante que crestate e indintate undas P amonta insimul a solmente 14 pro cento.

4. Ergo, a parte le differente circumstantias anatomic e functional, le frequentia cardiac debe esser prendite in consideration como un del factores fundamental que determina le forma del unda P.

REFERENCES

1. Pardee, H. E. B.: Clinical Aspects of the Electrocardiogram, ed. 4, New York, 1941, Paul B. Hoeber, Inc., p. 35.
2. Scherf, D., and Boyd, L. J.: Clinical Electrocardiography, ed. 2, Philadelphia, 1946, J. B. Lippincott Company, p. 216.
3. Graybiel, A., McFarland, R. A., Gates, D. C., and Webster, F. A.: Analysis of the Electrocardiogram Obtained From 1000 Young Healthy Aviators, AM. HEART J. **27**:524, 1944.
4. Stewart, C. B., and Manning, G. W.: A Detailed Analysis of the Electrocardiograms of 500 R.C.A.F. Aircrew, AM. Heart J. **27**:502, 1944.
5. Lewis, T., and Gilder, M. D. D.: The Human Electrocardiogram; A Preliminary Investigation of Young Male Adults to Form a Basis for Pathological Study, Phil. Tr., Lond. B **202**:351, 1912.
6. Ashman, R., and Hull, E.: Essentials of Electrocardiography, New York, 1941, The MacMillan Company.
7. Shipley, R. A., and Hallaran, W. R.: The Four-Lead Electrocardiogram in Two Hundred Normal Men and Women, AM. HEART J. **11**:325, 1936.
8. Winternitz, M.: Zur Pathologhie des menschlichen Vorhofelektrokardiogramm, Med. Klin. **31**:1575, 1935.
9. Rothberger, C. J., and Scherf, D.: Zur Kenntnis der Erregungsausbreitung von Sinusknoten auf den Vorhof, Ztschr. ges. exp. Med. **53**:792, 1926.
10. Rothberger, C. J., and Winterberg, H.: Über die Beziehungen der Herznerven zur Form des Elektrokardiogramms, Arch. ges. Physiol. **135**:506, 1910.
11. Weller, J. M., Lown, B., Hoigne, R. V., Wyatt, N. F., Cristiello, M., Merril, J. P., and Levine, S. A.: Effects of Acute Removal of Potassium From Dogs. Changes in the Electrocardiogram, Circulation **11**:44, 1955.
12. Martin, H. E., and Wertman, M.: Electrolyte Changes and the Electrocardiogram in Diabetic Acidosis, AM. HEART J. **42**:671, 1951.
13. Surawicz, B., and Lepeschkin, E.: The Electrocardiographic Pattern of Hyperpotassemia With or Without Hypocalcemia, Circulation **8**:801, 1953.
14. Sodi-Pallares, D., and Marsico, F.: The Importance of Electrocardiographic Patterns in Congenital Heart Disease, AM. HEART J. **49**:202, 1955.

MUSCULAR CONTRACTION IN THE INFUNDIBULAR REGION AS A MECHANISM OF PULMONIC STENOSIS IN MAN

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TWO significantly different types of infundibular pulmonic stenosis were demonstrated by blood pressure tracings recorded during right heart catheterization in man. Such a differentiation may have importance in the surgical approach to these malformations. The mechanisms involved may also be responsible, in part, for the bidirectional shunting of blood through associated ventricular septal defects.

MATERIAL AND METHODS

The records of thirteen patients found to have infundibular pulmonic stenosis† form the basis of the present report (cf. Table I). The ages ranged from 4 to 24 years. Studies were made during a reasonably steady resting state, some of the younger patients being under rectal Pentothal anesthesia.

A cardiac catheter was passed through an antecubital vein to the pulmonary artery; pressures and blood samples were obtained as the catheter was withdrawn in stages. Pressures were recorded by a capacitance manometer on a direct-writing polyoscillograph (Sanborn). Blood samples were analyzed for oxygen content by the method of Van Slyke and Neill.

Representative tracings from the main pulmonary artery, infundibulum, and right ventricle were superimposed on the same time base using the peak of the R of the electrocardiogram as a zero point of reference.

Certain potential sources of error were taken into consideration. These included: (1) the very slight changes in the level of the catheter tip with respect to the zero level of the manometer, (2) recording of pressures through a catheter which passes through the beating heart, (3) the recording of the tracing at the several sites consecutively, rather than simultaneously, (4) errors associated with

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†This condition is diagnosed on catheterization by the drop in systolic pressure between the right ventricle and a "chamber" proximal to the pulmonary artery.

plotting of the tracings, (5) partial occlusion of the infundibular stenosis by the shaft of the catheter when its tip is beyond the stenosis, and (6) artefacts due to temporary occlusion of the catheter opening. The magnitude of these errors was not considered to be sufficient to affect the validity of the results.

TABLE I. SUMMARY OF DATA

CASE	FIG.	AGE AND SEX	PRESSURES (mm. Hg)				PER CENT OF TOTAL PULMONARY FLOW DUE TO LEFT-TO-RIGHT SHUNT	PER CENT OF OXYGEN SATURATION SYSTEMIC ARTERY
			RIGHT VENTRICLE	INFUNDIBULUM	PULMONARY ARTERY	SYSTEMIC ARTERY		
Synchronous Group								
L.G.	1,*A*	M 10	110/5	70/5	25/10*	127/55	0	87
J.H.	1,*B*	F 11	35/0	18/0	18/8	110/70	0	?
J.W.		F 4	90/0	7/0	7/3	90/55	25	56
Asynchronous Group								
D. Po.	2,*A*	F 13	55/0	20/0	20/5	90/55	0	98
E.C.	2,*B*	M 24	82/0	70/0	10/2*	95/55	32	90
C.G.	2,*C*	F 11	90/2	38/2	18/5*	105/68	42	97
D.Ph.		M 10	110/10	50/10	50/20	95/60	60	95
D.M.		M 6	90/0	25/0	25/10	80/60	45	96
H.H.		F 23	90/4	25/4	12/6*	90/60†	33	84
H.Wo.		F 9	90/0	25/0	25/6	90/60†	39	94
R.T.	2,*D*	M 9	70/2	20/2	20/7	100/57	32	97
M.G.	2,*E*	F 7	105/0	15/0	12/4	75/55†	0	89
H.We.		M 10	100/3	25/3	17/6*	75/58	28	88

*Additional valvular pulmonic stenosis.
†Aorta.

RESULTS

The pertinent hemodynamic data are summarized in Table I.

Right ventricular and infundibular pressures were identical in each of the thirteen cases during the brief interval between the commencement of ventricular systole and the onset of the pressure rise in the pulmonary artery (isometric contraction of the right ventricle). The pressure relationships between right ventricular and infundibular curves during the remainder of ventricular systole made possible the division of the cases into two distinct groups.

1. *Synchronous Group.*—In three instances right venticular and infundibular systolic pressures rose and fell together, the peaks being simultaneous. In the two cases illustrated (Fig. 1,*A* and *B*) the slopes of the ascending limbs of the two pressure curves were actually coincident during the early part of ventricular ejection. The slope of the ascending limb of the infundibular pressure curve then decreased with the appearance of a pressure gradient between the two tracings. This gradient was most marked at the time of the simultaneous peaks. Following this the ventriculo-infundibular (V-I) gradient decreased

progressively, and the two curves became coincident again for a period prior to isometric relaxation. The third case was similar except that the pressure gradient was apparent throughout the period of ventricular ejection.

2. *Asynchronous Group.*—In ten cases the infundibular pressure reached its peak significantly ahead of the peak pressure in the right ventricle (Fig. 2). Early in the period of maximal ejection, the two pressure curves have very similar slopes and, in some cases, segments of considerable length could be superimposed exactly (Fig. 2,*A* and *B*). The infundibular pressure reached its peak within an average of 0.09 second (± 0.03) after the onset of systole, and then declined sharply although right ventricular pressure continued to rise. This more pronounced gradient late in systole contrasts this group with the synchronous group. The slope of the two curves did not again become coincident until the period of isometric relaxation.

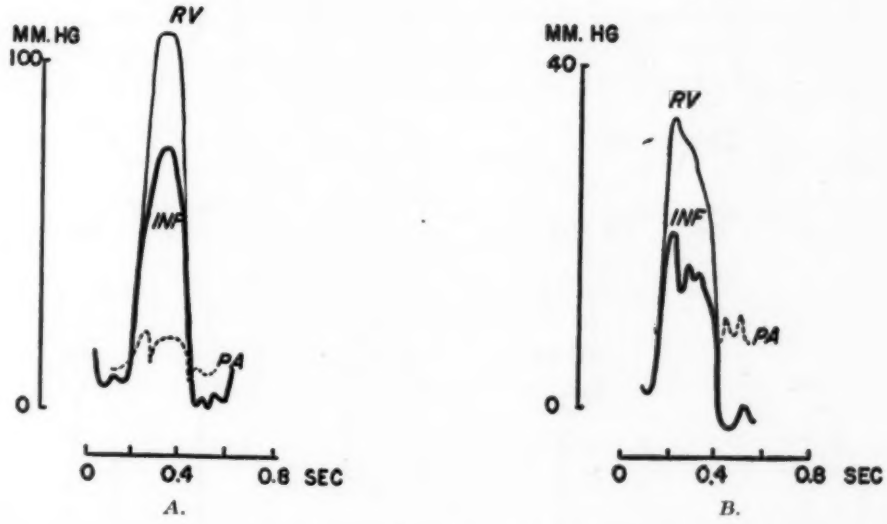

Fig. 1.—Superposition of pressure pulses from the right ventricle (*RV*, solid light line), the infundibulum (*INF*, solid heavy line) and pulmonary artery (*PA*, broken light line). Pressures are given in mm. Hg on the vertical axis. Time is given in seconds on the horizontal axis. *A* is case L. G., having valvular pulmonic stenosis in addition; *B* is case J. H. The synchrony of the systolic pressure peaks of right ventricle and infundibulum suggests that these cases represent instances of a fixed stenotic band in the infundibular region. Note that the diastolic portions of the infundibular and right ventricular curves correspond in *A* and *B*, while the systolic portions of the infundibular and pulmonary arterial curves correspond in *B*. The infundibular curve of *B* is distorted by catheter motion artifact in the latter part of its summit.

The absence of a V-I gradient until very near the peak of the infundibular pressure tracing indicated that there was no apparent resistance at this site to right ventricular ejection early in systole. This was found in three of the ten cases of the asynchronous group, two of which are illustrated in Fig. 2,*A* and *B*.

In four other cases, the ascending limbs of the infundibular and right ventricular systolic pulses had identical slopes at the onset of systolic ejection, as in the preceding, but some time before the peak of the infundibular curve was reached, the slope became less steep than that of the right ventricle (Fig. 2,*C*). Here,

infundibular stenosis was manifested by a V-I gradient early in systole, but only at higher rates of flow. Again, the gradient between the two chambers was much more marked in later systole.

In the remaining three cases the V-I gradient was apparent throughout systolic ejection, becoming more marked in later systole (Fig. 2,*D* and *E*).

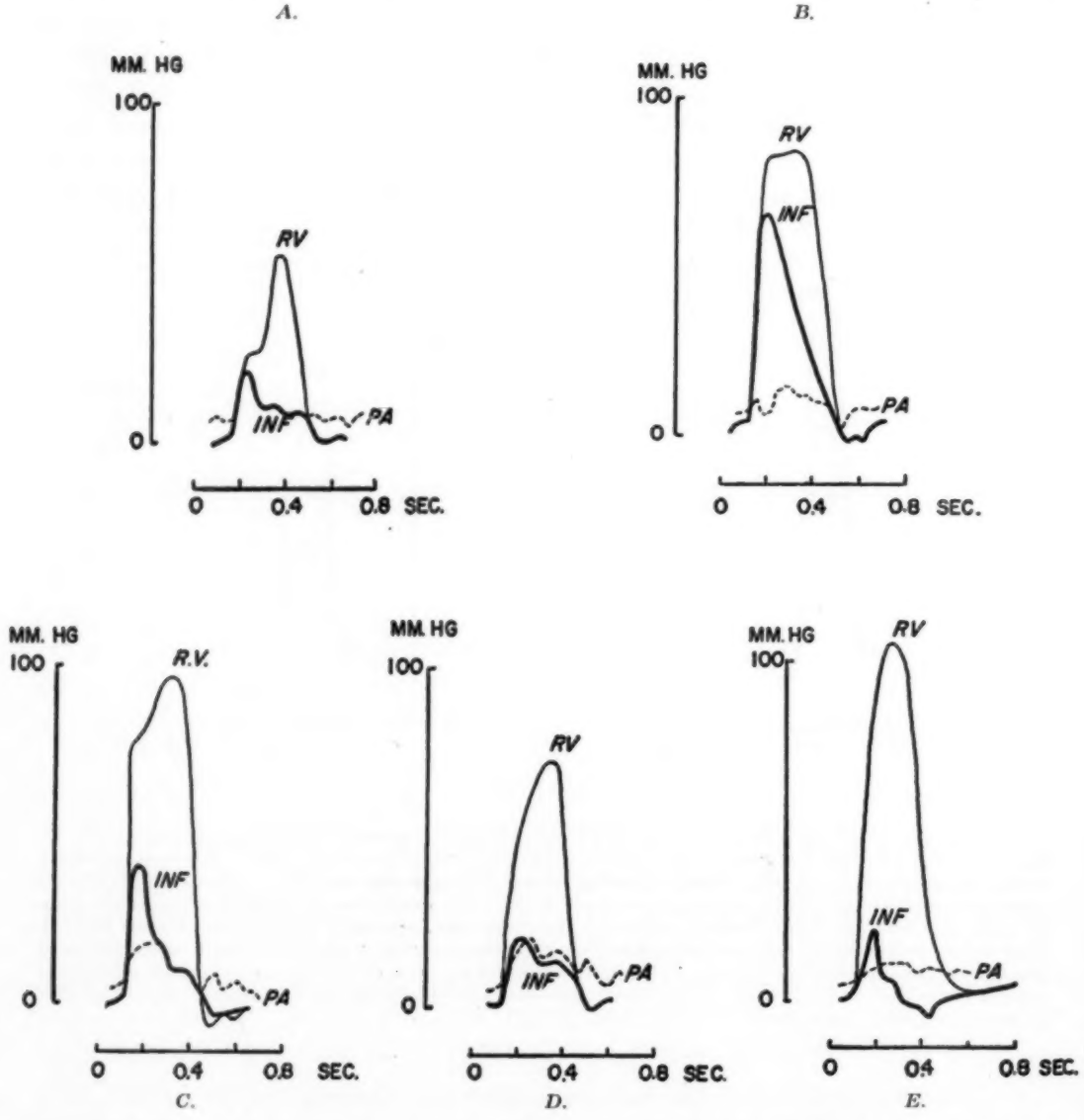

Fig. 2.—Pressure tracings in five cases illustrating asynchronism of the systolic pressure peaks of the infundibulum and right ventricle. Conventions as in Fig. 1. *A*, *B*, *C*, *D*, and *E* represent, respectively, cases D. Po., E. C., C. G., R. T., and M. G. Cases E. C. and C. G. had valvular pulmonic stenosis in addition. In *E*, the infundibular pressure curve falls below that of the pulmonary artery in late systole, probably due to a "Venturi" effect. Note that the diastolic portions of the infundibular and right ventricular curves correspond closely in all cases, while the systolic portions of the infundibular and pulmonary arterial curves correspond in *A* and *D*. Although the apparent interruption in the upstroke of the ventricular curve in *A* may be artifact, it may possibly represent right ventricular contraction tending toward the isometric as the outflow tract narrows in systole.

In this subgroup the V-I gradient over all of systole was generally the largest in the series.

The case illustrated in Fig. 2,*E* is of particular interest since the infundibular malformation could be evaluated at surgery. In addition, the pulmonary arterial pressure exceeded infundibular pressure during the latter part of ventricular systole.

DISCUSSION

Right ventricular and infundibular pressure tracings embodying the features analyzed in this study are not uncommon in the literature,[1-6] but their significance has not been previously commented upon.

During flow through a fixed stenotic orifice the pressures on either side will vary in the same direction, rising and falling together, and any peaks and troughs will be virtually simultaneous. Here, the pressure gradient varies only with the rate of flow through the stenotic opening. Thus, with a mild fixed infundibular stenosis, not manifest by a pressure gradient at low rates of flow the right ventricular and infundibular pressure tracings may bear a relationship as in Fig. 3,*A*.

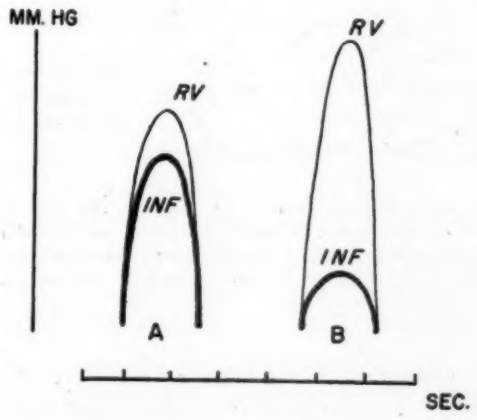

Fig. 3.—A schematic representation of simultaneous pressure curves in the right ventricle (*RV*) and infundibulum (*INF*), with (*A*) mild fixed infundibular stenosis and (*B*) severe fixed infundibular stenosis. Conventions as in Fig. 1. In both instances the peaks of the ventricular and infundibular curves are reached simultaneously. In *A*, the lesser degree of stenosis is manifested by the congruency of the two pressure curves at low rates of flow while a gradient is present at higher rates of flow, as in mid-systole. In *B* a gradient is present throughout the period of ventricular ejection.

A more severe fixed stenosis may produce a gradient at any significant rate of flow as in Fig. 3,*B*. Experimentally produced fixed stenosis[7] and instances of valvular pulmonic stenosis (personal observation), in which the stenosis would seem to be relatively fixed on anatomic grounds, resulted in similar pressure relationships across the stenosed orifice. In the present study the synchronous group was characterized by such a relationship between the infundibular and right ventricular pressure tracings, suggesting that the stenotic orifice of the infundibulum was of constant area throughout systole.

No pressure gradient was observed between right ventricle and infundibulum during the period of isometric contraction in any of our cases. Since this is a period of zero flow between two chambers in equilibrium, no gradient is to be expected regardless of any narrowing between them.

In the asynchronous group, the infundibular pressure curve, during ejection, was skewed relative to that of the right ventricle. Regardless of the magnitude of the V-I gradient early in systole, it became relatively marked later in systole, disappearing only at the onset of isometric relaxation. Barring artifacts, this can be explained only by the assumption that changes in the cross section of the infundibular orifice took place during systole.

Although the mechanism producing this change in calibre of the stenotic channel has not yet been demonstrated in man, there is indirect evidence strongly supporting the hypothesis that it is due to myocardial contraction in the region of the infundibulum some time after the onset of systole. Phylogenetic evidence (Fig. 4) and observations on the turtle heart[9,10] indicate that the pulmonary conus is one of the last portions of the myocardium to contract during systole, and this may be true in man, as suggested by electrocardiographic observations.[11]

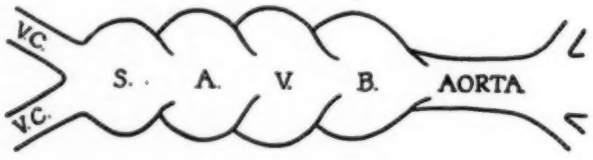

Fig. 4.—A diagram of the primitive heart. At the left, the venae cavae (*VC*) enter the sinus venosus (*S*). This chamber contracts at the onset of systole and blood is moved to the atrium (*A*), the ventricle (*V*), and the bulbus (*B*), which is the last part of the heart to contract. The bulbus gives rise to the infundibulum of the right ventricle in man. See also reference 8.

The peak of the infundibular pressure curve, occurring about a tenth of a second after the commencement of ventricular systole, could mark the time in the cycle when active contraction of the myocardium in the region of the conus begins to manifest itself by an effect on the pressure gradient, this effect persisting until the end of systole. The tracings of the asynchronous group indicate that muscular contraction in this region may be the sole cause of infundibular stenosis (Fig. 2,*A* and *B*). Such contraction may also aggravate a stenosis demonstrable, to a varying extent, with the infundibulum in the relaxed state (Fig. 2,*C*, *D*, and *E*). At any rate, the cyclic V-I gradient is thus the resultant of two variable factors—the dimensions of the outflow tract and the rate of right ventricular ejection—neither of which can be quantitated. For this reason, calculation of cross-sectional area of such a stenotic orifice based on standard hydraulic formulae can have only limited meaning.

The pathologic anatomy of infundibular pulmonic stenosis has been extensively described.[12-15] In brief there are two varieties. One takes the form of a fibrous band at the site of the normal constriction between the cavities of the

right ventricle and of the infundibulum. In the second type, the cavity of the infundibulum itself is shrunken and "tunnel-like" due to a distorted and abnormally large muscle mass (the crista supraventricularis). It would be useful from a practical point of view to differentiate these two types, since a fibrous band presents a much less formidable surgical problem than does a muscular tunnel.

This study suggests a possible relationship between the anatomy and the hemodynamics of infundibular pulmonic stenosis based on the hypothesis that further narrowing in the infundibular stenosis is due to myocardial contraction in that region. We have been able to check this in the case illustrated in Fig. 2,*E*.

The cross-section of a "band" stenosis is likely to remain fixed during ventricular systole, while that of a "tunnel" stenosis would, in all likelihood, be decreased. The latter effect may be accentuated by the greater length of a "tunnel." The sequence in the synchronous group indicating a fixed infundibular stenosis is thus more likely to be of the "band" type. In the asynchronous group, by contrast, a muscle component may be operating, and the stenosis is more likely to be of the "tunnel" variety. The fact that ten of the thirteen cases in our series are of the "tunnel" type suggests that this is not an uncommon mechanism of infundibular stenosis.

The mechanism in the asynchronous group which causes the infundibular pressure to fall off before the peak of ventricular systole must be distinguished from that underlying the systolic dip in tracings recorded just distal to a severe valvular pulmonic stenosis.[16,17] The dip can be accounted for on the basis of the high velocity of blood flow through a narrowed orifice manifesting itself as a local fall in lateral pressure. The lowest point of a systolic dip is synchronous with the highest point on the right ventricular curve or the latter is slightly earlier. In the asynchronous group of the present series, the premature fall in infundibular pressure during systole can be ruled out as a manifestation of this "Venturi" effect because the pulmonary arterial pressure curve conforms in level and contour to the infundibular curve, or is lower due to valvular pulmonic stenosis. The case illustrated in Fig. 2,*E* is a possible exception because the pulmonary arterial pressure consistently exceeds the pressure in the infundibulum during late systole. The lowest point in the infundibular curve occurs 0.06 second after the peak of ventricular systole. This delay may be related to the late systolic accentuation of the "Venturi" phenomenon by the further narrowing of the infundibular canal.

The differences in resistance to outflow produced by constriction of the infundibulum during early systole in the asynchronous group probably contribute to the cyclic fluctuations in the direction and volume of flow across the adjacent ventricular septal defect. Thus, from Fig. 2, the left-to-right shunt demonstrated in eight of ten cases in the asynchronous group is most likely to occur early in systole, and to decrease or reverse as the resistence to flow into the pulmonary artery increases because of the more marked stenosis later in systole.

This impression of the effects of infundibular contraction is fortified by observations on the heart of the turtle which has a large ventricular septal defect as part of its normal anatomy. In this species a ring of muscle below the

pulmonary valve may sometimes be observed to commence contraction in mid-systole, narrowing the pulmonary outlet. This has been shown to play an important role in maintaining systemic flow at the expense of pulmonary flow under certain conditions.[8]

This analysis indicates that careful scrutiny of the pressure tracings obtained in the right ventricle and in the infundibulum can provide considerable insight into the nature of the stenotic lesion between these chambers.

SUMMARY

Two discrete types of infundibular pulmonic stenosis were differentiated by an analysis of records of cardiac catheterizations in thirteen patients. In three of these, the right ventricular and infundibular pressure tracings had the same phase relationships, rising together, having simultaneous peaks, and falling together. Since the peaks occurred simultaneously it was assumed that the stenotic orifice between the two chambers had a fixed area. This would correspond to the type of infundibular stenosis characterized anatomically by a fibrous band between right ventricle and infundibulum.

In the remaining ten cases, the pressures in the two chambers began to rise together, but the ascending limb of the infundibular pressure tracing was cut off prematurely during early systole. Then the right ventricular pressure continued to rise while the infundibular pressure fell. The peaks of the two curves were, therefore, asynchronous. The pressure gradient between the two curves in this group was wider in late than in early systole. It is postulated that the stenosed opening is narrowed or further narrowed as a result of contraction of a muscular ring during early systole. This group may correspond to those with an enlarged or deformed crista supraventricularis. The presence of such a muscular ring was confirmed at surgery in one patient in this group. Analysis of the pressure tracings provided a basis for bidirectional shunting across associated ventricular septal defects.

SUMMARIO IN INTERLINGUA

Duo discrete typos de stenose pulmonic infundibular esseva differentiate per un analyse del protocollos de catheterisation cardiac in dece-tres patientes. In tres del casos, le registrationes del pression dextero-ventricular e del pression infundibular habeva le mesme relationes phasic: illos ascendeva insimul, habeva crestas simultanee, e descendeva insimul. Proque le crestas occurreva simultaneemente, nos concludeva que le orificio stenotic inter le duo cameras habeva un area fixe. Isto corresponderea al typo de stenose infundibular que es characterisate anatomicamente per un banda fibrose inter le ventriculo dextere e le infundibulo.

In le remanente dece casos, le pressiones in le duo cameras comenciava lor ascendita insimul, sed le membro ascendente del curva de pression infundibular esseva abrumpite prematuremente in le comenciamento del systole. Postea le pression dextero-ventricular continuava ascender durante que le pression infundibular descendeva. Ergo le crestas del duo curvas esseva asynchome.

Le gradiente de pression inter le duo curvas in iste gruppo de casos esseva plus grande al termino que al initio del systole. Nos postula que le apertura stenotic es restringite (o restringite additionalmente) como resultato del contraction de un anulo muscular al initio del systole. Iste gruppo es possibilemente characterisate per un allargamento o un distorsion del cresta supraventricular. Le presentia de un tal anulo muscular esseva confirmate al operation in un patiente de iste gruppo. Le analyse de registrationes de pression provideva un base pro le supposition del presentia de un derivation bidirectional a transverso associate defectos ventriculo-septal.

We are indebted to the members of the catheterization team for their assistance in obtaining the data in these patients, and to Dr. Louis N. Katz for valuable discussions during the development of this study.

REFERENCES

1. Mannheimer, E. (editor): Morbus Coeruleus: An Analysis of 114 Cases of Congenital Heart Disease With Cyanosis, Basel, 1949, S. Karger.
2. Dow, J. W., Levine, H. D., Elkin, M., Haynes, F. W., Hellems, H. K., Whittenberger, J. W., Ferris, B. G., Goodale, W. T., Harvey, W. P., Eppinger, E. C., and Dexter, L.: Studies of Congenital Heart Disease. IV. Uncomplicated Pulmonic Stenosis, Circulation **1**:267, 1950.
3. van Buchem, F. S. P., Nieveen, J., Homan, B., and Verhey, J. B.: Rétrécissement isolé de l'artère pulmonaire, Cardiologia **19**:248, 1951.
4. Soulié, P., and others: Les cardiopathies congénitales, Paris, 1952, L'Expansion scientifique française.
5. Bayer, O., Loogen, F., Wolter, H. H.: Der Herzkatheterismus bei Angeborenen und Erworben Herzfehler, Stuttgart, 1954, Georg Thieme.
6. McCord, M. C., and Blount, S. G., Jr.: Complications Following Infundibular Resection in Fallot's Tetralogy, Circulation **11**:754, 1955.
7. Katz, L. N., Ralli, E. P., and Cheer, S. N.: The Cardiodynamic Changes in the Aorta and Left Ventricle Due to Stenosis of the Aorta, J. Clin. Invest. **5**:205, 1927.
8. Clark, A. J.: Comparative Physiology of the Heart, London, 1927, Cambridge University Press.
9. Woodbury, R. A., and Robertson, G. G.: The One Ventricle Pump and the Pulmonary Arterial Pressure of the Turtle: The Influence of Artificial Acceleration of the Heart, Changes in Temperature, Hemorrhage and Epinephrine, Am. J. Physiol. **137**:628, 1942.
10. Rodbard, S., Brown, F., and Katz, L. N.: The Pulmonary Arterial Pressure, Am. Heart J. **38**:863, 1949.
11. Kossmann, C. E., Berger, A. R., Radner, B., Brumlik, J., Briller, S. A., and Donnelly, J. H.: Intracardiac and Intravascular Potentials Resulting From Electrical Activity of the Normal Human Heart, Circulation **2**:10, 1950.
12. Keith, A.: The Hunterian Lectures on Malformations of the Heart, Lecture I, Lancet **1**:359, 1909.
13. Baffes, T. G., Johnson, F. R., Potts, W. J., and Gibson, S.: Anatomic Variations in the Tetralogy of Fallot, Am. Heart J. **46**:657, 1953.
14. Johns, T. N. P., Williams, G. R., and Blalock, A.: The Anatomy of Pulmonary Stenosis and Atresia With Comments on Surgical Therapy, Surgery **33**:161, 1953.
15. Lev, M.: Autopsy Diagnosis of Congenitally Malformed Hearts, Springfield, Ill., 1953, Charles C Thomas.
16. Sobin, S. S., Carson, M. J., Johnson, J. L., and Baker, C. R.: Pulmonary Valvular Stenosis With Intact Ventricular Septum: Isolated Valvular Stenosis and Valvular Stenosis Associated With Interatrial Shunt, Am. Heart J. **48**:416, 1954.
17. Bouchard, F., and Cornu, C.: Etude des courbes de pressions ventriculaire droite et artèrielle pulmonaire dans les rétrécissements pulmonaires, Arch. mal. coeur **47**:417, 1954.

A RADIAL DEVICE FOR THE RLF PLANE

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SÃO PAULO, BRAZIL

IN CLINICAL electrocardiography Einthoven and associates[1] assumed that the three limb electrodes were equidistant from each other and from the heart, the interposed tissue was homogeneous in conductivity, and the heart was a point source of electromotive forces. Consequently they proposed an equilateral triangle for calculating cardiac vectors in the RLF plane. Based upon this geometric figure several devices have been constructed which are believed to be useful in routine use. Among these may be quoted the charts of Carter and associates[2] and Dieuaide,[3] the triaxial reference system of Bayley,[4] the hexaxial reference system of Sodi-Pallares and associates,[5] and the polarity circle system of Zao and associates.[6-11]

In present communication a radial device constructed on the same basis for calculating cardiac vectorial directions will be given. It will be discussed that a similar device may be constructed on the basis of a Burger triangle.[12]

DESCRIPTION AND USE

This device (Fig. 1) contains seventy-two radii, each being separated 5 degrees from the neighboring one and bears a quotient, e_1/e_3, as will be described. Each radius indicates an Einthoven direction. The polarity circle of L_I divides this device into a left half and a right half. (Each polarity circle consists of one positive semicircle, one negative semicircle, and two zero potential demarcations between them.) The left positive semicircle is colorless; the right negative semicircle is gray.

Let e_1 = any deflection in L_I, mean, main, or instantaneous
e_3 = corresponding deflection in L_{III}.

To use this device one visualizes at first the polarity of e_1. If it is positive, the vectorial direction is within the left positive semicircle; if it is negative, the direction is within the right negative semicircle. (If e_1 is zero, the vectorial direction points toward $+90°$ when e_3 is positive, toward $-90°$ when e_3 is negative.) Then the quotient e_1/e_3 will be calculated and correlated with a radius which bears the most proximate value. This radius indicates the given vectorial direction.

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CONSTRUCTION

Fig. 2 shows the rudiment of this device. $\hat{E}$, $\hat{E}'$ = projections of two oppositely directed cardiac vectors in the RLF plane.

Let e_1, e_3 = projections of $\hat{E}$ on L_I, L_{III}
e_1', e_3' = projections of $\hat{E}'$ on L_I, L_{III}
a, b = angles that $\hat{E}$ defines with L_I, L_{III}
a′, b′ = angles that $\hat{E}'$ defines with L_I, L_{III}.

Since a = a′, b = b′, one gets

$$e_1/e_3 = e_1'/e_3' = \cos a/\cos b. \quad (1)$$

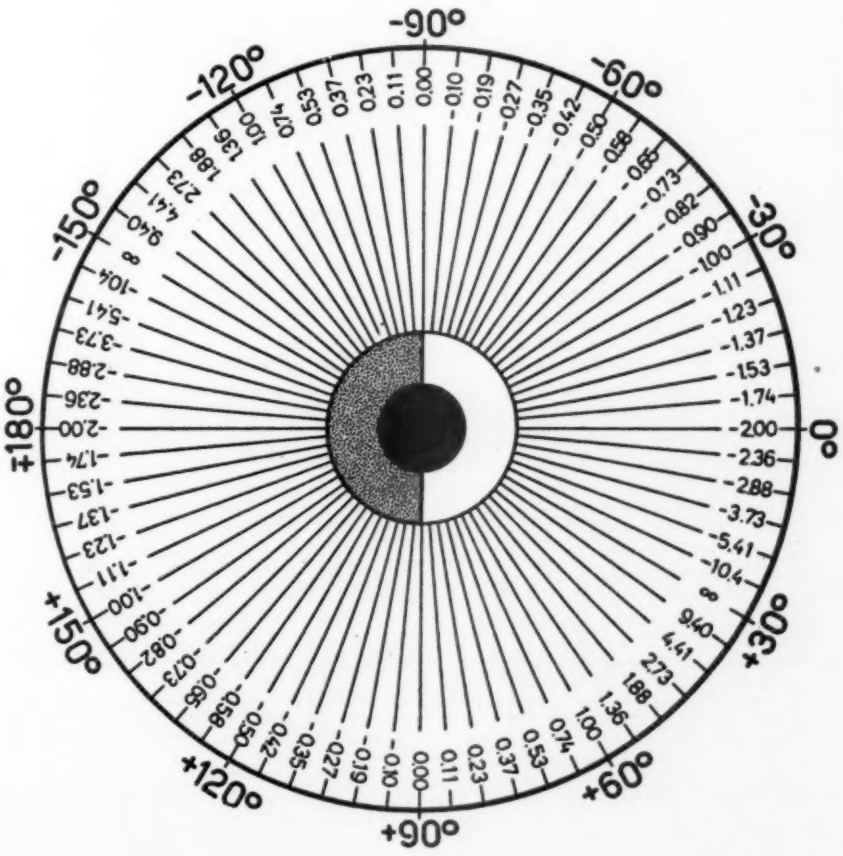

Fig. 1.—A radial device of the RLF plane.

If $\hat{E}$ and $\hat{E}'$ rotate counterclockwisely as indicated by the arrow, $\hat{E}$ will inscribe the left half of the RLF plane and $\hat{E}'$ the right half. At any instant the above equation holds true. The results from 5-degree intervals were listed in Table I.

It was also made as a graphic approach, using $\hat{E}$ of 10 cm. length in each instance, and measured directly e_1, e_3 with a ruler. Results were identical.

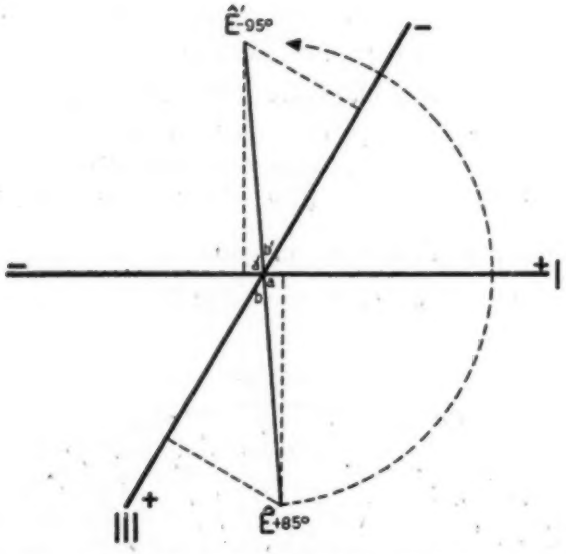

Fig. 2.—The rudiment of the radial device.

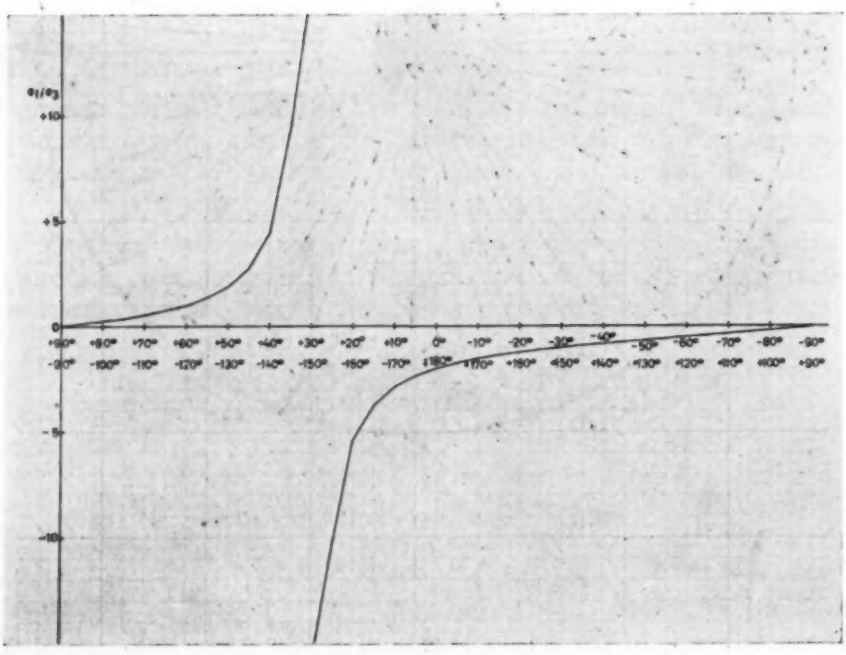

Fig. 3.—The two curves correlate quotients to Einthoven directions. Quotients were indicated on ordinate: positive above *O*, negative below *O*. On abscissae Einthoven directions were given: those from left half above, from right half below.

TABLE I

EINTHOVEN DIRECTIONS		COS A : COS B*	NATURAL TRIGONOMETRIC FUNCTIONS	$E_1 : E_3$
LEFT HALF	RIGHT HALF			
+85°	− 95°	+cos 85° +cos 35°	+0.08716:+0.81915	+0.10640297
+80°	−100°	+cos 80° +cos 40°	+0.17365:+0.76604	+0.2266852
+75°	−105°	+cos 75° +cos 45°	+0.25882:+0.70771	+0.3660250
+70°	−110°	+cos 70° +cos 50°	+0.34202:+0.64279	+0.5320866
+65°	−115°	+cos 65° +cos 55°	+0.42262:+0.57356	+0.7368109
+60°	−120°	+cos 60° +cos 60°	+0.50000:+0.50000	+1.0000000
+55°	−125°	+cos 55° +cos 65°	+0.57358:+0.42262	+1.3572003
+50°	−130°	+cos 50° +cos 70°	+0.64279:+0.34202	+1.8793930
+45°	−135°	+cos 45° +cos 75°	+0.70711:+0.25882	+2.7320531
+40°	−140°	+cos 40° +cos 80°	+0.76604:+0.17365	+4.4114022
+35°	−145°	+cos 35° +cos 85°	+0.81915:+0.08716	+9.3982330
+30°	−150°	+cos 30° cos 90°	+0.86603: 0.00000	∞
+25°	−155°	+cos 25° −cos 85°	+0.90631:−0.08716	−10.3982330
+20°	−160°	+cos 20° −cos 80°	+0.93969:−0.17365	−5.4114022
+15°	−165°	+cos 15° −cos 75°	+0.96595:−0.25882	−3.7320531
+10°	−170°	+cos 10° −cos 70°	+0.98481:−0.34202	−2.8793930
+ 5°	−175°	+cos 5° −cos 65°	+0.99619:−0.42262	−2.3571766
0°	∓180°	+cos 0° −cos 60°	+1.00000:−0.50000	−2.0000000
− 5°	+175°	+cos 5° −cos 55°	+0.99619:−0.57358	−1.7367934
−10°	+170°	+cos 10° −cos 50°	+0.98481:−0.64279	−1.5322535
−15°	+165°	+cos 15° −cos 45°	+0.96593:−0.70711	−1.3660250
−20°	+160°	+cos 20° −cos 40°	+0.93969:−0.76604	−1.2266852
−25°	+155°	+cos 25° −cos 35°	+0.90631:−0.81915	−1.1064029
−30°	+150°	+cos 30° −cos 30°	+0.86603:−0.86603	−1.0000000
−35°	+145°	+cos 35° −cos 25°	+0.81915:−0.90631	−0.9038298
−40°	+140°	+cos 40° −cos 20°	+0.76604:−0.93969	−0.8152050
−45°	+135°	+cos 45° −cos 15°	+0.70771:−0.96593	−0.7320509
−50°	+130°	+cos 50° −cos 10°	+0.64279:−0.98481	−0.6527045
−55°	+125°	+cos 55° −cos 5°	+0.57358:−0.99619	−0.5757736
−60°	+120°	+cos 60° −cos 0°	+0.50000:−1.00000	−0.5000000
−65°	+115°	+cos 65° −cos 5°	+0.42262:−0.99619	−0.4242363
−70°	+110°	+cos 70° −cos 10°	+0.34202:−0.98481	−0.3472954
−75°	+105°	+cos 75° −cos 15°	+0.25882:−0.96593	−0.2679490
−80°	+100°	+cos 80° −cos 20°	+0.17365:−0.93969	−0.1847949
−85°	+ 95°	+cos 85° −cos 25°	+0.08716:−0.90631	−0.09617018

*The sign preceding each cos indicates the polarity of the lead side when $\hat{E}$ inscribes the left half of the RLF plane.

DISCUSSION

Any pair of limb leads may be used to construct an RLF plane radial device. If a bipolar limb lead is to be paired with a unipolar one, a standardizing factor must be considered.[13] Leads I and III were chosen arbitrarily.

The quotient direction relationship may be presented in curve form, as shown in Fig. 3. Using the same criteria, it can be correlated exactly. But the curve form seems to be less lucid in routine use.

However, the three limb electrodes are not equidistant from each other, the human heart is not an electric point source and it locates excentrically toward L electrode, and the interposed tissue is nonhomogeneous. Thus the Einthoven ideal conditions are not consistent with reality. His triangle and subsequent devices derived from it cannot give accurate results no matter how

exactly they are used.[14,19] Nevertheless, the Einthoven triangle has the support of clinical empiricism of more than forty years. It would be very interesting to know to what extent this empiricism is inaccurate.

On the other hand, a Burger triangle gives almost accurate results. Because its construction takes real conditions, such as thorax form, eccentricity of the heart, and tissue nonhomogeneity into account. Subsequent studies[15-22] suggest that this triangle may prove more valuable in clinical electrocardiography than that of Einthoven, though its form may not yet be accepted as being definitive.[19]

To use such a triangle it is necessary before beginning the usual procedure to divide the deflections written in limb leads by the length of the corresponding sides. Theoretically a Burger triangle may be transformed into an axial reference figure with proper divisions on each side, which serves immediately the usual procedure.[23] This has been tested satisfactorily on subsequent experiments.[22] Using such a figure, the quotient e_1/e_3 may be obtained graphically at present. The results may be superimposed in the present device.[24] It may be anticipated that with which the interpreter will get simultaneously two cardiac vectorial directions in the RLF plane from each quotient, the empirical one from the Einthoven triangle and the accurate one from the Burger triangle. His diagnosis will be more secure. Conversely, if a vectorial direction from the Einthoven triangle is known and its e_1/e_3 quotient is indicated, the accurate direction from the Burger triangle can be found. In this way the inaccuracy of the Einthoven triangle in clinical empiricism of over forty years may be tested.

SUMMARY

A radial device was given on the basis of the Einthoven triangle. Using the quotient e_1/e_3 the corresponding cardiac vectorial direction in the RLF plane can be obtained. It was discussed that a similar device could be constructed on the basis of a Burger triangle. The same quotient could give almost the accurate direction.

SUMMARIO IN INTERLINGUA

Un construction radial esseva elaborate super le base del triangulo de Einthoven. Per usar le quotiente e_1/e_3, on pote obtener le correspondente direction del vector cardiac in le plano RLF. Es signalate que un simile construction es possibile super le base del triangulo de Burger. Le mesme quotiente resultarea in le determination quasi exacte del direction del vector cardiac.

REFERENCES

1. Einthoven, W., Fahr, G., and de Waart, A.: Über die Richtung und manifeste Grösse der Potentialschwankungen im menschlichen Herzen und über den Einfluss der Herzlage auf die Form des Elektrokardiogramms, Pflügers Arch. ges. Physiol. **150**:275, 1913. English translation, AM. HEART J. **40**:163, 1950.
2. Carter, E. P., Richter, C. P., and Greene, C. H.: A Graphic Application of the Principle of the Equilateral Triangle for Determining the Direction of the Electrical Axis of the Heart in the Human Electrocardiogram, Bull. Johns Hopkins Hosp. **30**:162, 1919.
3. Dieuaide, F. R.: The Determination and Significance of the Electrical Axis of the Human Heart, Arch. Int. Med. **27**:558, 1921.

4. Ashman, R., and Byer, E.: The Normal Human Ventricular Gradient. I. Factors Which Affect Its Direction and Its Relation to the Mean QRS Axis; With an Appendix on Notation, By R. H. Bayley, AM. HEART J. **25**:16, 1943.
5. Sodi-Pallares, D., Cuéllar, A., and Cabrera, E.: Sistema de seis ejes con aplicación al vector AvT en las hipertrofias ventriculares, Arch. Inst. cardiol. México **14**:142, 1945.
6. Zao, Z. Z., and Laranja, F. S.: Sistema hexaxial com círculos de polaridade: um método simples para determinação da direção dos vetôres cardíacos no plano frontal; descrição geral do método, Arq. brasil. cardiol. **5**:82, 1952.
7. Zao, Z. Z., and Laranja, F. S.: Método simples para eletro-vector-cardiografia espacial, O Hospital **43**:735, 1953.
8. Zao, Z. Z., and Laranja, F. S.: Técnica para construção do vetorcardiograma espacial pelo sistema de polaridades a partir das derivações eletrocardiográficas usuais, Rev. argent. cardiol. **19**:287, 1952.
9. Zao, Z. Z.: A utilidade do sistema de referência circular das derivações das extremida des na rotina clínica, Arq. brasil. cardiol. **7**:7, 1954.
10. Zao, Z. Z.: Estudo quantitativo do ÂQRS, ÂT e ângulo ÂQRS-ÂT no plano frontal em traçados normais e com hipertrofia ventricular; analise de 4189 traçados, Presented at the Sixth Annual Meeting, Brazilian Association for the Advancement of Science, 1954.
11. Zao, Z. Z.: The Two Main QRS Vectors in the Frontal Plane in Electrocardiograms of Complete Right Bundle Branch Block, Cardiologia (in press).
12. Burger, H. C., and van Milaan, J. B.: Heart Vector and Leads. I., Brit. Heart J. **8**:157, 1946.
12a. Burger, H. C., and van Milaan, J. B.: Heart Vector and Leads. II., Brit. Heart J. **9**:154, 1947.
13. Cronvich, J. A., Conway, J. P., and Burch, G. E.: Standardizing Factors in Electrocardiography, Circulation **2**:111, 1950.
14. Zao, Z. Z.: Burger Triangle as a Method for Correcting Inaccuracies of Einthoven Triangle, Science **122**:375, 1955.
15. Wilson, F. N., Bryant, J. M., and Johnston, F. D.: On the Possibility of Constructing an Einthoven Triangle for a Given Subject, AM. HEART J. **37**:493, 1949.
16. den Boer, W.: Vectorcardiographie (a Thesis), Utrecht, 1951, Rijks Universiteit.
17. Becking, A. G. T., Burger, H. C., and van Milaan, J. B.: Universal Vector Cardiograph, Brit. Heart J. **12**:339, 1950.
18. McFee, R., Stow, R. M., and Johnston, F. D.: Graphic Representation of Electrocardiographic Leads by Means of Fluid Mappers, Circulation **6**:21, 1952.
19. Brody, D. A., and Romans, W. E.: A Model Which Demonstrates the Quantitative Relationship Between the Electromotive Forces of the Heart and the Extremity Leads, AM. HEART J. **45**:263, 1953.
20. Zao, Z. Z.: Beitrag zum Burgerschen Dreieckschema, Ztschr. f. Kreislaufforsch. **44**:593, 1955.
21. Helm, R. A.: Theory of Vectorcardiography: A Review of Fundamental Concepts, AM. HEART J. **49**:135, 1955.
22. Zao, Z. Z.: An Experimental Study of Burger Triangles Constructed From Toad Hearts in Situ, Circulation Research (in press).
23. Brody, D. A.: The Meaning of Lead Vectors and the Burger Triangle, AM. HEART J. **48**:730, 1954.
24. Zao, Z. Z.: A Device Which Gives Simultaneous Results From Einthoven Triangle and Burger Triangle, In Preparation.

THE DURATION OF ATRIAL ELECTRICAL ACTIVITY AND ITS RELATIONSHIP TO THE ATRIAL RATE

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THE existence of a T_P wave as part of the atrial complex was first described by Hering in 1912.[1] The P wave is believed to represent atrial excitation and the T_P wave is believed to represent atrial recovery. Study of the T_P wave is difficult because most of it is buried in the deflection resulting from ventricular activity. The entire atrial electrical complex ($P + T_P$) can only be clearly distinguished in the presence of atrioventricular block.

It is well known that deviations of the S-T junction and segment may be due to the T_P wave. In recognition of this, the Committee on Electrocardiography of the American Heart Association has recommended that the end of the P-R segment be used as the level of reference for determining S-T junction deviations and for measuring the voltage of the QRS deflections.[2]

The duration of the P wave has been determined in a small group of cases and has been shown to increase with decreasing atrial rate.[3] Except for several isolated measurements, the $P + T_P$ duration has not been studied. Sprague and White measured the $P + T_P$ duration in five cases with complete heart block.[4] Battro and Bidoggia measured the $P + T_P$ duration in an intracardiac lead in one patient with "A-V block."[5] Brown measured seventeen $P + T_P$ durations in a study of esophageal electrocardiography and found the $P + T_P$ duration to vary from 0.405 to 0.503 second.[5a] Six $P + T_P$ durations were measured by Lambert.[6] Ashman and Hull[7] state that the $P + T_P$ duration varies inversely with heart rate and that the $P + T_P$ duration in the limb leads usually varies between 0.28 to 0.42 second.

In the course of a recent investigation of the spatial ventricular gradient, it was necessary to make correction for the effect of the T_P wave on the baseline used for measuring the areas of the QRS complex, the S-T segment, and the T wave.[8]

MATERIAL AND METHOD

The electrocardiographic records of thirty-six patients with different degrees of heart block were used in this study. These records were obtained on the wards and in the clinics of The Mount Sinai Hospital. Twenty-six patients

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had complete atrioventricular block and ten patients had incomplete atrioventricular block. Fifty determinations were made on the electrocardiograms obtained from thirty-five patients. One unusual patient (Case 36) with Adams-Stokes Syndrome due to complete atrioventricular block had numerous electrocardiograms from which twenty-four measurements of $P + T_P$ duration were made at various atrial rates. This interesting patient and his hospital course are being reported in detail elsewhere by Robbin and Dack.[9]

There were three records with atrial rates of 200 per minute or over. One had paroxysmal atrial tachycardia at a rate of 200 with 2:1 atrioventricular block. The two others had atrial flutter at rates of 248 and 300 per minute with 2:1 and 3:1 atrioventricular block, respectively. The latter two records were included in our measurements because each showed an isoelectric segment following the T_P wave.

In twenty-seven of the patients, arteriosclerotic heart disease was responsible for the atrioventricular block. One of the patients had acute rheumatic fever and complete atrioventricular block. Eight of the patients had congenital heart disease. Of the latter group, six patients with congenital complete atrioventricular block are the subjects of a separate study.[10]

The $P + T_P$ duration (in seconds) was measured directly from the original electrocardiographic records, most of which were taken at a paper speed of 50 mm. per second with a Technicon Cardiograph standardized so that 1 millivolt input produced an upright deflection of 15 mm. The P-P interval (in seconds) was similarly measured from the peaks of the appropriate adjacent P waves. The atrial rate (in cycles per minute) was then calculated by dividing 60 seconds by the P-P interval.

RESULTS

The data obtained are recorded in Table I. The table lists the age in years, the $P + T_P$ duration in seconds, the P-P interval in seconds, and the atrial rate in cycles per minute for each case measured. Fig. 1 is a graph in which the $P + T_P$ duration is plotted against the P-P interval and the atrial rate. The fifty points obtained from Cases 1 through 35 are denoted by dots and the twenty-four points obtained from Case 36 are denoted by crosses.

Three straight lines are fitted to the data by the method of least squares.[11] The line made up of long dashes (line A) is derived from the fifty points of Cases 1 through 35. The short-dashed line (line B) is derived from the twenty-four points of Case 36. The solid line (line C) is fitted to all seventy-four points. The equations for the three lines are: $(P + T_P) = 0.13 + 0.36(P\text{-}P)$ for line A, $(P + T_P) = 0.17 + 0.29(P\text{-}P)$ for line B, and $(P + T_P) = 0.14 + 0.34(P\text{-}P)$ for line C, where the $P + T_P$ duration and the P-P intervals are in seconds and $(P + T_P)$ and $(P\text{-}P)$ represent, respectively, the durations of $P + T_P$ and the P-P interval.

It is evident that the lines on the graph are extremely close to each other and that the line derived from measurements in one individual is in close agreement with the results derived from a group of different individuals.

TABLE I. RESULTS

NO.	AGE	P + TP (SECONDS)	P-P (SECONDS)	ATRIAL RATE (CYCLES/MIN.)	NO.	AGE	P+TP (SECONDS)	P-P (SECONDS)	ATRIAL RATE (CYCLES/MIN.)	NO.	AGE	P + TP (SECONDS)	P-P (SECONDS)	ATRIAL RATE (CYCLES/MIN.)
1	71	0.33	0.56	107	19	60	0.34	0.55	109	36	55	0.45	0.80	75
2	50	0.44	0.76	79	20	34	0.50	1.00	60			0.42	0.80	75
		0.40	0.72	83			0.44	0.90	67			0.38	0.77	78
		0.37	0.67	90			0.28	0.61	98			0.42	0.77	78
3	56	0.40	0.56	107	21	16	0.52	0.78	77			0.34	0.72	83
4	60	0.30	0.50	120	22	4 mos.	0.32	0.41	145			0.35	0.71	84
5	30	0.36	0.80	75	23	44	0.42	0.79	76			0.37	0.70	86
6	54	0.31	0.47	128	24	30	0.40	0.97	62			0.38	0.70	86
7	55	0.32	0.81	74	25	7	0.36	0.75	80			0.35	0.68	88
8	64	0.27	0.58	104			0.32	0.63	96			0.34	0.68	88
9	48	0.42	0.75	80			0.28	0.55	109			0.35	0.64	94
10	28	0.25	0.42	143			0.24	0.34	176			0.35	0.60	100
11	72	0.35	0.60	100	26	4	0.31	0.56	107			0.34	0.57	105
12	73	0.38	0.65	92	27	61	0.26	0.30	200*			0.36	0.57	105
13	56	0.38	0.60	100			0.19	0.20	300**			0.34	0.54	111
14	67	0.26	0.45	133	28	52	0.24	0.36	165			0.34	0.53	114
15	8	0.25	0.48	125	29	67	0.40	0.59	102			0.32	0.49	123
16	52	0.45	0.90	67			0.37	0.56	108			0.32	0.48	125
17	70	0.36	0.65	92	30	64	0.32	0.54	112			0.30	0.47	128
		0.34	0.62	97			0.32	0.51	117			0.31	0.43	137
		0.29	0.46	130	31	66	0.40	0.58	104			0.30	0.41	145
18	57	0.53	0.97	62	32	55	0.26	0.38	158			0.29	0.40	150
		0.46	0.84	71	33	80	0.36	0.76	79			0.27	0.38	158
		0.41	0.75	80	34	65	0.22	0.24	248**			0.26	0.36	167
		0.40	0.70	86	35	5	0.30	0.54	111					

Case 15 = complete A-V block with acute rheumatic fever; Cases 20 to 25 = complete A-V block with congenital heart disease; Cases 26 and 35 = Incomplete A-V block with congenital heart disease; Cases 27 to 34 = Incomplete A-V block with arteriosclerotic heart disease; All others = complete A-V block with arteriosclerotic heart disease.

***Paroxysmal atrial tachycardia.**

****Atrial flutter.**

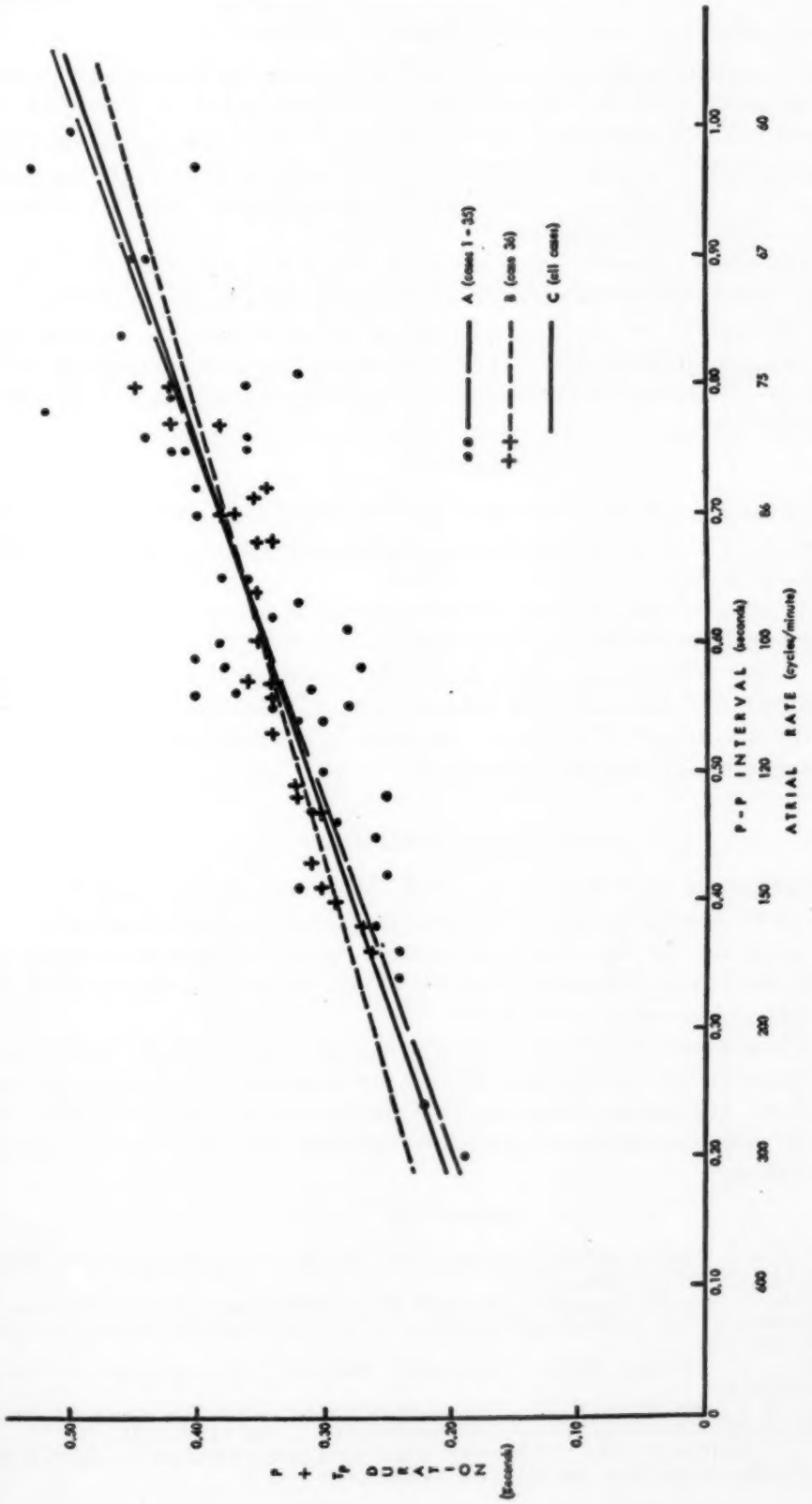

Fig. 1.—The relationship between the P + T_P duration and the P-P interval and the atrial rate. P + T_P duration in seconds is on the ordinate, and P-P interval in seconds, and atrial rate in cycles per minute are on the abscissa.

DISCUSSION

A linear relationship exists between the $P + T_P$ duration and the P-P interval within the limits for atrial rate of 60 to 300 cycles per minute. As the atrial rate increases and the P-P interval decreases, the $P + T_P$ duration decreases. This is a property common to several electrophysiologic events occurring in the heart. The P duration,[3] the QRS interval,[12] the P-R interval,[7] and the Q-T interval[7] decrease when heart rate increases.

To evaluate the degree to which the slope of our fitted straight line (line C) significantly differs from horizontal, having regard to the chance errors of random sampling, the standard error of the slope constant, 0.34, has been calculated.[11] The value obtained is 0.021, which is only one-sixteenth of the magnitude of the slope constant. The slope of the fitted straight line, therefore, significantly differs from the horizontal.

SUMMARY

1. The duration of atrial electrical activity, the P-P interval, and the atrial rate are measured in seventy-four electrocardiograms of thirty-six patients with atrioventricular block.

2. The range of the duration of atrial electrical activity ($P + T_P$) is 0.19 second at an atrial rate of 300, to 0.53 second at an atrial rate of 62.

3. A linear relationship exists between the duration of atrial electrical activity and the P-P interval with the duration of atrial electrical activity increasing with increasing P-P interval. Decrease in the duration of atrial electrical activity occurs with increase in atrial rate.

SUMMARIO IN INTERLINGUA

Le duration del electroactivitate atrial (P + Tp), le intervallo P-P, e le frequentia atrial esseva mesurate in septanta-quatro electrocardiogrammas ab trenta-sex patientes con bloco atrioventricular. Le limites minimo-maximal del duration del electroactivitate atrial esseva 0,19 sec con un frequentia atrial de 300 e 0,53 sec con un frequentia atrial de 62.

Esseva constatate le existentia de un relation linear inter le duration del electroactivitate atrial e le intervallo P-P: le duration del electroactivitate atrial se augmentava quando le intervallo P-P monstrava un augmentation. Un reduction del duration del electroactivitate atrial occurre quando le frequentia atrial se augmenta.

REFERENCES

1. Hering, H. E.: Über die Finalschwanking (Ta-Zache) des Vorhafelektrogramms, Pflüger's Arch. ges. Physiol. **144**:1, 1912.
2. Committee on Electrocardiography, American Heart Association: Recommendations for Standardization of Electrocardiographic and Vectorcardiographic Leads, Circulation **10**:564, 1954.
3. Lepeschkin, E.: Modern Electrocardiography, Baltimore, 1951, Williams & Wilkins Company.
4. Sprague, H. B., and White, P. D.: Clinical Observations in the T Wave of the Auricle Appearing in the Human Electrocardiogram, J. Clin. Invest. **1**:389, 1925.
5. Battro, A,, and Bidoggia, H.: Endocardiac Electrocardiogram Obtained by Heart Catheterization in the Man, Am. Heart J. **33**:604, 1947.

5a. Brown, W. H.: A Study of the Esophageal Lead in Clinical Electrocardiography: Part I. The Application of the Esophageal Lead to the Human Subject With Observations in the T_A Wave, Extrasystoles, and Bundle Branch Block, AM. HEART J. **12**:1, 1936.
6. Lambert, J.: Le decalage combine des segments PR et RT de l'electrocardiogramme. Signification clinique, Acta cardiol. **2**:37, 1947.
7. Ashman, R., and Hall, E.: Essentials of Electrocardiography, New York, 1941, The Macmillan Company.
8. Berkun, M. A., Kesselman, R. H., Donoso, E., and Grishman, A.: The Spatial Ventricular Gradient: Intermittent Wolff-Parkinson-White Syndrome, Intermittent Left Bundle Branch Block, and Ventricular Premature Contractions, Circulation, In Press.
9. Robbin, S. R., and Dack, S.: The Treatment of Complete Heart Block With Isuprel. In Preparation.
10. Donoso, E., Braunwald, E., Jick, S., and Grishman, A.: Congenital Heart Block, Am. J. Med., In Press.
11. Pearl, R.: Introduction to Medical Biometry and Statistics. Philadelphia, 1941, W. B. Saunders Company.
12. Lepeschkin, E., and Surawicz, B.: The Duration of the Q-V Interval and Its Components in Electrocardiograms of Normal Persons, AM. HEART J. **46**:9, 1953.

ELECTROCARDIOGRAM'S DISTORTIONS DUE TO A HIGH SKIN-ELECTRODE RESISTANCE

A Simple Clinical Method for Measuring This Resistance

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SINCE the introduction of amplifier electrocardiograph, scant attention has been paid to the effect of skin-electrode resistance on the form and amplitude of the electrocardiographic deflections; this attitude has been largely due to the common belief that the high input resistance of these apparatus makes the skin resistance unimportant.

In recent years many reports have called attention to the effect of inequality of contact resistance at each limb on the potential of the central terminal,[1-6] which is shifted toward the electrode with the lower resistance. The errors so introduced are more noticeable in either augmented or nonaugmented unipolar limb leads and may be ascribed to a shift in the axis of the unipolar leads clockwise or counterclockwise to a maximum of 30 degrees to either side of the correct direction.[7] This effect is minimized by the use of 5,000 ohm resistors in the Wilson central terminal, provided that the skin resistance in each limb is kept sufficiently low.

With regard to errors introduced in standard limb leads and in unipolar precordial leads by a high skin resistance when using an amplifier electrocardiograph, they have until now received little attention. In a recent paper[8] the author has stressed the importance of adequate preparation of the patient's skin, especially when the input resistance of the electrocardiograph is as low as 200,000 ohms (such as in a well-known battery-operated apparatus) or even 68,000 ohms (such as in a three-channel modern instrument[9]). A high contact resistance affects the electrocardiogram in two ways: (1) It diminishes the amplitude of the electrocardiographic waves; (2) It introduces distortions in these waves through skin and electrode capacity. These errors are proportional to the ratio of the skin and electrode resistance to the total circuit resistance[10] including the input resistance of the apparatus, so that care in the preparation of the skin could be safely disregarded only when using machines with input resistance of several megohms. As this is not always the case, special care must be taken to ensure an adequate preparation of the patient's skin through the use of a suitable electrode paste.

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In any case, a method for measuring the skin resistance at each electrode seems highly desirable. Goldberger[11] has described such a method, but it is only applicable to string galvanometer electrocardiographs, and Jeanneret[12] has described another one suitable for the amplifier type of apparatus involving the use of a dry cell and a voltage divider. In the present paper a simple method is described which makes it possible to measure the electrical resistance under any electrode by means of an amplifier electrocardiograph and a 5,000 or 10,000 ohm resistor as the only additional component.

PRINCIPLE

The method is the same as used in electronics for measuring the output impedance of audio-frequency amplifiers at a given frequency. The circuit diagram is shown in Fig. 1,*A*. *Zi* is the internal unknown impedance of the

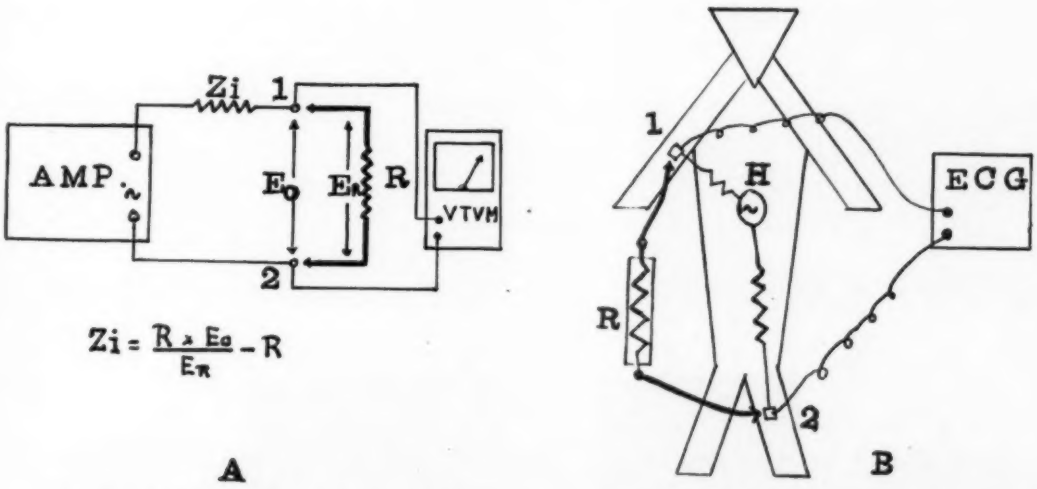


Fig. 1.—*A*, Diagram circuit for measuring the output impedance of an audiofrequency amplifier. *B*, Arrangement of the electrical circuit for measuring body resistance. A 5,000 ohm resistor is connected across the electrode lead wires being utilized.

amplifier (*AMP.*). The open circuit voltage (*Eo*) at its terminals *1* and *2* is measured by means of a vacuum tube voltmeter (*VTVM*) with a high input impedance. If we connect the terminals *1* and *2* by a resistor (*R*) of a known value, the voltage at these terminals will be now E_R, smaller than *Eo*. The value of *Zi* may be then calculated by the following formula:

$$Zi = \frac{R \times Eo}{E_R} - R \qquad (1)$$

In Fig. 1,*B*, the amplifier is substituted by the heart (generating low frequency waves), the terminals *1* and *2* are the corresponding electrodes of any of the electrocardiographic leads, and the VTVM is replaced by an amplifier electrocardiograph. The open circuit voltage (Eo) is now obtained by careful

measuring of the amplitude of QRS deflections to the nearest two-tenths of a millimeter in the lead recorded in the normal manner. Then a resistor R is connected in parallel across the corresponding electrode wires and the lead is newly registered. QRS deflections, now diminished in amplitude, are measured as E_R. After appropiate correction for errors in standardization have been made, if necessary, the value of the body resistance at this lead will be given by equation (1). In practice, the two wire leads of the resistor are supplied with clips at its ends in order to facilitate its rapid connection. Five thousand to ten thousand ohms are satisfactory values for the resistor R.

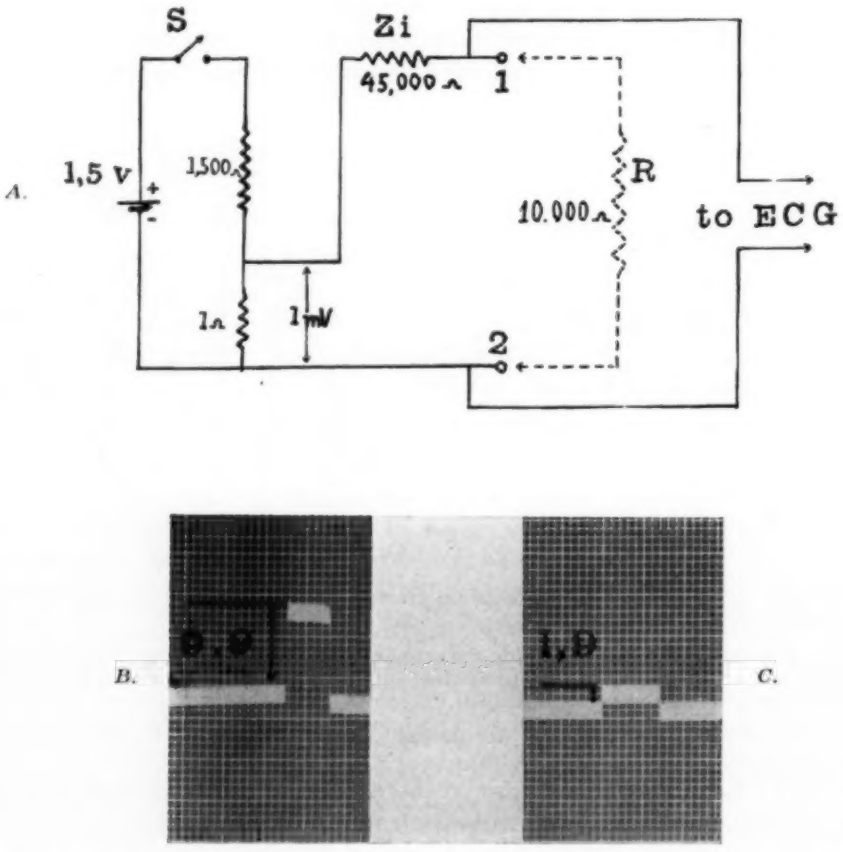


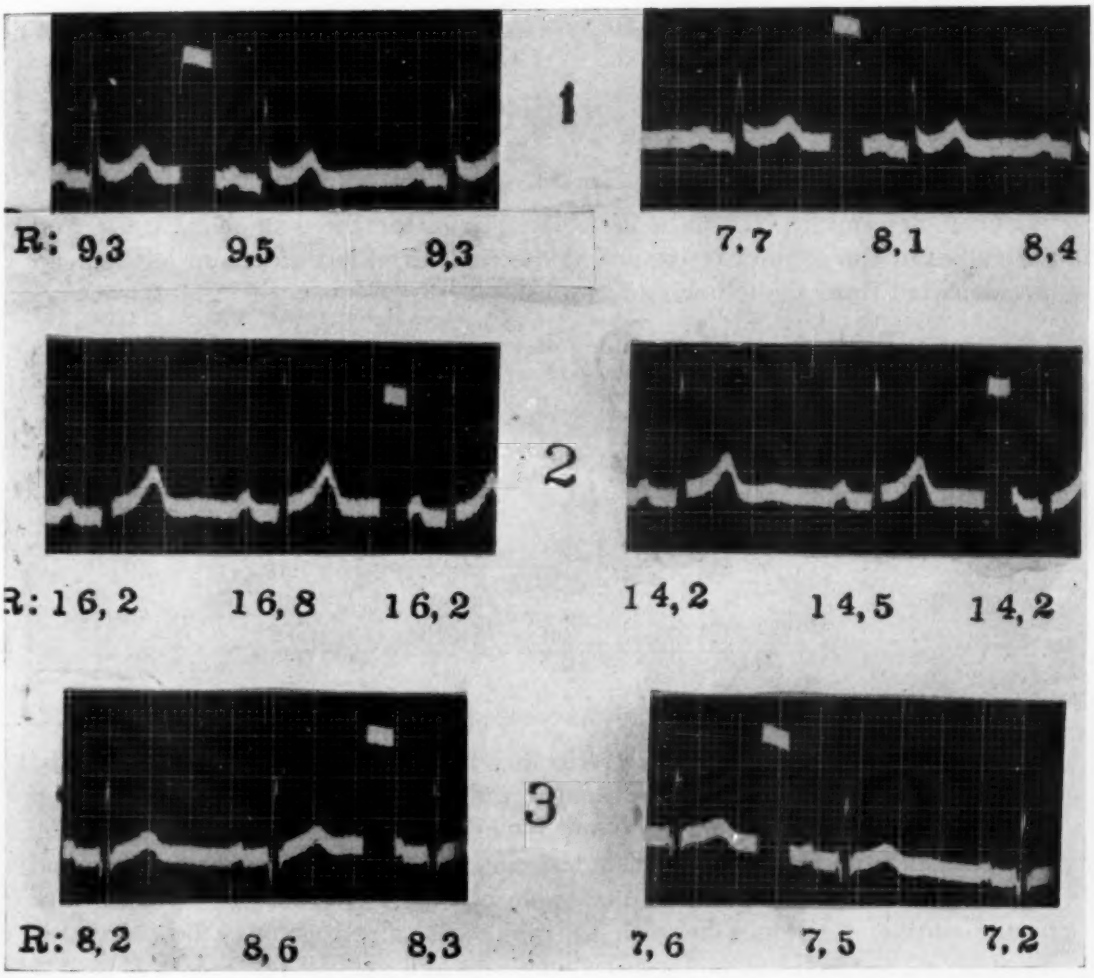
Fig. 2.—*A*, Circuit used to check the method. *B*, Open circuit rectangular wave. *C*, Rectangular wave obtained after connecting a 10,000 ohm resistor (*R*) across terminals *1* and *2*.

In order to test experimentally the accuracy of this method, we have used the circuit shown in Fig. 2,*A*, in which a dry cell of 1.5 volts, in series with a voltage divider provides 1 millivolt at the terminals of the 1 ohm resistor. *Zi*, of a known value, represents the internal resistance of the signal input source. A switch (*S*) supplies rectangular waves which are registered by the electrocardiograph. Fig. 2,*B* shows the open circuit rectangular waves thus obtained,

measuring 9.9 mm. in height. When a 10,000 ohm resistor (R) is connected across terminals 1 and 2, the amplitude of the output pulse is reduced to 1.9 mm. (Fig. 2,*C*). According to the equation (1):

$$Zi = \frac{10{,}000 \times 9.9}{1.9} - 10{,}000 = 42{,}100 \text{ ohms.}$$

This result is roughly concordant with the known value of Zi (45,000 ohms).



A. *B.*

Fig. 3.—Lead I, II, and III obtained before (*A*) and after (*B*) connecting a 5,000 ohm resistor. See text for details.

Fig. 3 shows the application of the procedure in a practical case. In Fig. 3,*A* are reproduced Leads I, II, and III which were registered in the usual manner. In Fig. 3,*B* the same leads were taken after connecting successively a 5,000 ohm resistor in parallel across each lead being registered. The amplitude of R wave in each pair of records was carefully measured and averaged from three beats. The values listed in Table I were then obtained, applying equation (1).

TABLE I. LEAD RESISTANCE VALUES AS OBTAINED FROM FIG. 3,*A* AND *B*

LEAD	AMPLITUDE OF R WAVE WITHOUT SHUNT WITH A RESISTOR	AMPLITUDE OF R WAVE AFTER CONNECTING A 5,000 OHM RESISTOR	LEAD RESISTANCE (OHMS)
I	$\frac{28.1}{3} = 9.3$	$\frac{24.2}{3} = 8.6$	$1,610 = R_1$
II	$\frac{49.2}{3} = 16.4$	$\frac{42.9}{3} = 14.3$	$1,440 = R_2$
III	$\frac{25.1}{3} = 8.36$	$\frac{22.3}{3} = 7.43$	$1,300 = R_3$

Once determined, the patient's resistance in the three standard leads, and the values of the contact resistance at the right arm, left arm, and left leg can be calculated from the following formulas[11]:

$$R_{RA} = \frac{R_1 + R_2 - R_3}{2}; \quad R_{LA} = \frac{R_1 + R_3 - R_2}{2}; \quad R_{LL} = \frac{R_2 + R_3 - R_1}{2}$$

In this particular case:

$$R_{RA} = \frac{1,610 + 1,440 - 1,300}{2} = 875 \text{ ohms}$$

$$R_{LA} = \frac{1,610 + 1,300 - 1,440}{2} = 735 \text{ ohms}$$

$$R_{LL} = \frac{1,440 + 1,300 - 1,610}{2} = 565 \text{ ohms}$$

All these values are graphically presented in Fig. 4.

In a similar way the contact resistance at any precordial electrode can be determined. In this case one terminal of the 5,000 or 10,000 ohm resistor must be attached to the central terminal and the other one to the precordial electrode. By recording the precordial lead before and after connecting the resistor and applying equation (1) the resistance value of this lead is obtained. The value of the contact resistance beneath the precordial electrode may be calculated by applying the well-known formulas for series and parallel resistances (Fig. 5,*A* and *B*):

$$R_{PE} = \frac{1}{\frac{1}{R_{RA} + R_{CT}} + \frac{1}{R_{LA} + R_{CT}} + \frac{1}{R_{LL} + R_{CT}}}$$

where R_{PE} = resistance beneath the precordial electrode,
R_{PL} = resistance value of the precordial lead,
R_{RA}, R_{LA}, and R_{LL} = contact resistance at right arm, left arm, and left leg, respectively, and
R_{CT} = fixed resistors used in Wilson central terminal (usually 5,000 ohms).

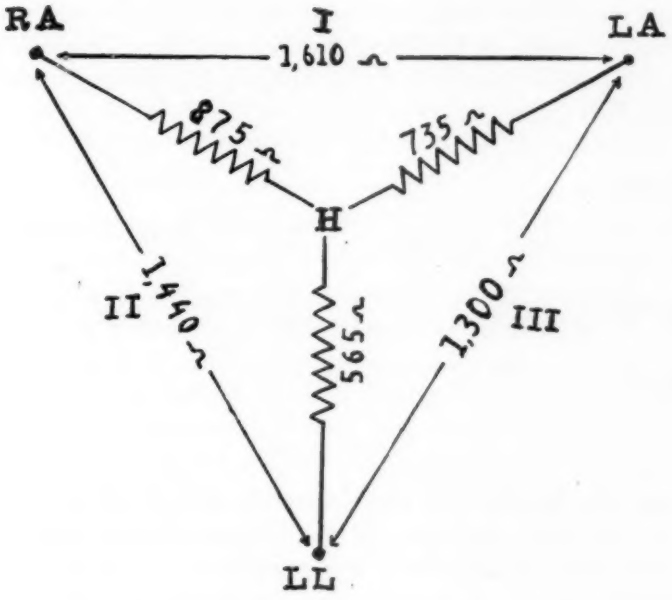


Fig. 4.—Resistance values obtained from Fig. 3.

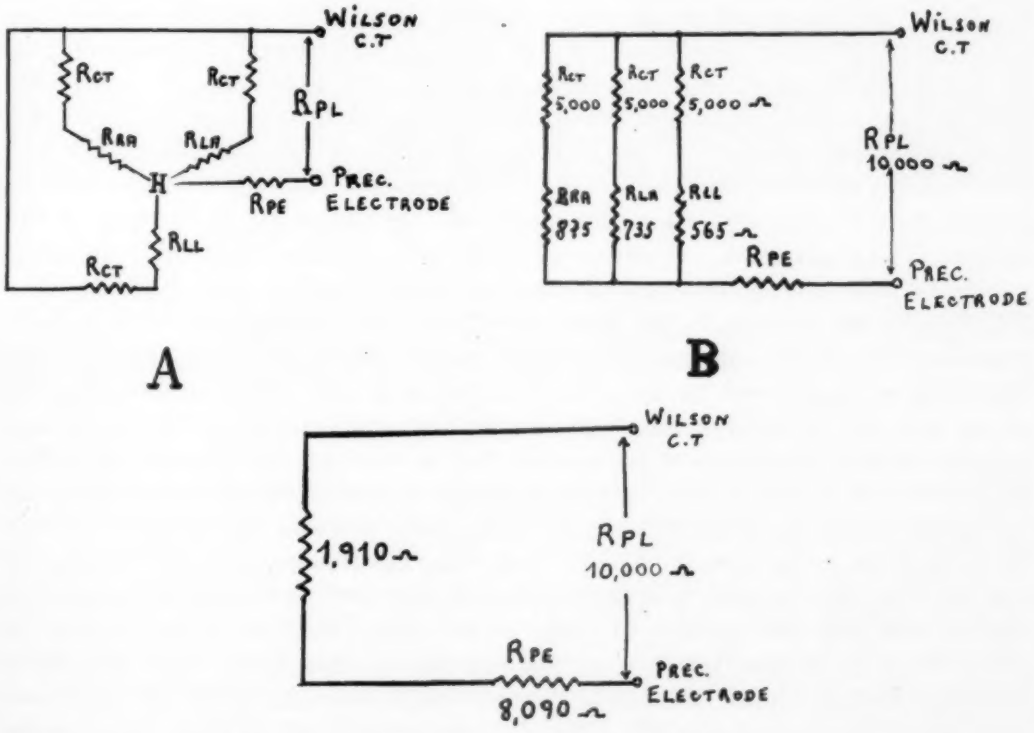


Fig. 5.—*A*, Circuit diagram of the precordial leads. *B*, Rearrangement of circuit diagram shown in *A*. *C*, Equivalent circuit of *B*.

As an example, we can assume that in the particular case above mentioned we have obtained a value of 10,000 ohms in Lead V_1. The other values were: 5,000 ohms for R_{CT}, 875 ohms for the right arm, 735 ohms for the left arm, and 565 ohms for the left leg. Then:

$$R_{V_1} = 10{,}000 - \frac{1}{\frac{1}{875 + 5.000} + \frac{1}{735 + 5.000} + \frac{1}{565 + 5{,}000}}$$

$$= 10{,}000 - \frac{1}{0.000170 + 0.000173 + 0.000179}$$

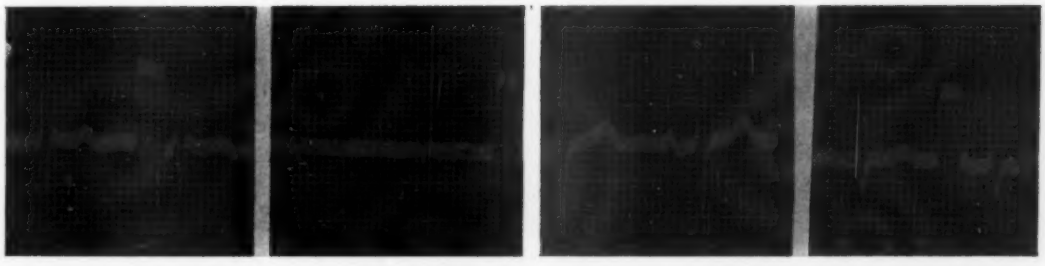
$$= 10{,}000 - 1{,}910 = 8{,}090 \text{ ohms}$$

DISCUSSION

It is not our purpose here to make an exhaustive study on the value of body resistance under several conditions of health and disease, but rather to give a simple clinical method for measuring this resistance. In fact, we have made one or more measurements in a large number of individuals, varying the skin preparation and the electrode paste. We found that with a poor electrode jelly and improper preparation of the patient's skin, the contact resistance may attain values as high as 17,000 ohms under limb electrodes and 48,000 ohms under precordial electrodes. Furthermore, the dissimilarity in contact resistance at the right arm, left arm, and left leg was greater in these cases than in those with lower values. Thus, in a case, these values were: 4,900 ohms for the right arm, 2,900 for the left arm, and 17,100 ohms for the left leg. Tracings obtained under these conditions will show: (1) a shift of the potential of the central terminal toward the electrode with the lower resistance, independently of the type of the electrocardiograph used. Insertion of 5,000 ohm resistors will be insufficient to correct this unbalanced central terminal; only resistors of a higher value, 50,000 ohms for instance, would mask this effect; (2) a decrease of the amplitude and distortion of the electrocardiographic waves; these last errors are due, the first (loss of amplitude) to the fact that the large skin-electrode resistance is acting as a voltage divider, thus reducing the voltage presented to the apparatus, and the second (distortion of the wave form) to the fact that the electrocardiographic circuit is not a pure resistance circuit but includes skin and electrode capacities which are parallel with the skin and electrode resistances.[10] When these resistances become unusually high, the electrocardiographic waves are distorted by the charging and discharging of the skin and electrode capacities during and after the passage of these waves, respectively.[8] These effects are partly dependent upon the type of the instrument used, being more noticeable in those with lower input resistance. Leads with greatest skin-electrode resistance and largest electrocardiographic deflections are more prone to show these errors. These conditions are usually fulfilled in precordial leads V_1 and V_2, where we have found resistance values as high as 48,000 and 60,000 ohms. In fact, the distortion due to a high skin resistance-low input impedence electrocardiograph may be recognized at first glance, in any tracing, by inspecting Leads V_1 or V_2:

there will be found a marked positive RS-T deviation arising from an apparent R′ wave and sloping gradually downward toward a T wave of diminished amplitude and with a final negative phase. Fig. 6,*A* shows such a distorted V_2 (lead resistance = 72,000 ohms) and Fig. 6,*B* shows the same lead obtained with a suitable conducting paste and proper preparation of the skin (lead resistance = 11,000 ohms).

Indeed, electrocardiograms containing errors of this nature have been frequently reproduced in textbooks and articles concerning cardiology. For instance, in a recent paper Edeiken[13] describes elevation of RS-T segment in the right precordial leads as a probable normal variant. Inspection of tracings corresponding to Cases 2, 3, 5, 7, and 9 arouses the strong suspicion that this elevation was, at least in part, a technical artifact, due to skin and electrode capacitance and low input impedance of the apparatus used. Correct standardization square wave is not sufficient to discard this condition if the 1 millivolt potential is not inserted into each of the limb and chest leads, but directly injected into the input circuit of the machine.



A. *A′.* *B.* *B′.*

Fig. 6.—Lead V_1 registered with improper preparation of the skin (*A*). Distortion is evident as compared with the same lead obtained properly (*B*). *A′* and *B′* are the corresponding tracings obtained after connecting a 10,000 ohm resistor in parallel across the lead. Resistance values calculated by means of equation (1) were 72,000 and 11,000 ohms, respectively. Input impedance of the electrocardiograph used: 200,000 ohms.

With a proper technique involving a suitable conducting paste, clean electrodes, and rubbing of the skin with alcohol, we have obtained contact resistance values in limb electrodes ranging from 275 to 2,755 ohms. It is worthy of note that often these values were not strictly comparable for the same individual at different times and even for the same occasion when using simultaneously a separate pair of electrodes in each extremity. Thus, in a case, the values obtained for the proximal and distal electrodes were, respectively: 1,060 and 402 ohms for the right arm, 558 and 470 ohms for the left arm, and 430 and 1,132 for the left leg. These variations are not surprising in view of the many variable factors that influence the skin-electrode resistance (site and preparation of the skin, amount of the electrode jelly, pressure of the electrode, etc.). In any case they are immaterial with respect to the object pursued in this investigation.

SUMMARY

A high skin-electrode resistance affects the electrocardiogram in two ways: (1) producing an unbalanced central terminal through dissimilar contact in each of the three limb leads, and (2) giving distorted waves in any lead through skin and electrode capacitance. This last error is proportional to the ratio of the skin-electrode resistance to the total circuit resistance. Hence it is more common when using string electrocardiographs, but it is also found in those amplifier instruments which have low input impedance. Usually, the distorted waves are more noticeable in precordial Leads V_1 and V_2 and may be recognized in many tracings reproduced in papers and textbooks concerning cardiology. For these reasons, a clinical method for measuring the actual skin resistance seems highly desirable. Such a method, applicable to any type of electronic apparatus and requiring a 5,000 or 10,000 ohm resistor as the only additional component, is described. Formulas are given which allow the determination of skin resistance under any limb or precordial electrode.

SUMMARIO IN INTERLINGUA

Un alte resistentia de contacto cutaneo-electrodic affice le electrocardiogramma in duo manieras. (1) Illo produce un disbalanciate terminal central in consequentia de dissimile contactos del tres derivationes extremitatal. (2) Illo produce distortiones del undas in omne derivation per le reactantia de capacitate de pelle e electrodo. Iste ultime error es proportional al quotiente del resistentia cutaneo-electrodic e del resistentia del circuito total. Ergo illo es plus commun in le uso de electrocardiographos con galvanometros a corda, sed illo se incontra etiam con instrumentos a amplificator que ha un basse impedantia de entrata. Usualmente le distortiones del undas es plus notabile in le derivationes precordial V_1 e V_2 e pote esser recognoscite in multe electrocardiogrammas reproducite in articulos e manuales cardiologic. Pro iste rationes, un methodo clinic pro le mesuration del ver resistentia cutanee pare esser un urgente desiderato. Nos describe un tal methodo que es applicabile a omne genere de apparato electronic e que require nulle componente additional excepte un resistentia de 5.000 o 10.000 ohms. Nos presenta formulas que permitte le determination del resistentia cutanee sub omne electrodo extremitatal o precordial.

REFERENCES

1. Bryant, J. M., Johnston, F. D., and Wilson, F. N.: Unipolar Electrocardiographic Leads. Effects Produced by Eliminating the Resistors Between the Limb Electrodes and the Central Terminal, Am. Heart J. **37**:321, 1949.
2. Rappaport, M. B., and Williams, C.: An Analysis of the Relative Accuracies of the Wilson and Goldberger Methods for Registering Unipolar and Augmented Unipolar Electrocardiographic Leads, Am. Heart J. **37**:892, 1949.
3. Kert, M. J., and Bryant, J. M.: The Potential Differences Between Multiple Central Terminals Each Connected to a Separated Set of Limb Electrodes, Am. Heart J. **37**:1035, 1949.
4. Cronwich, J. A., Conway, J. P., and Burch, G.: Standardizing Factors in Electrocardiography, Circulation **2**:111, 1950.
5. Langner, P. H., Malvern, Benjamin J., and Moore, S. R.: Certain Inevitable Relationship Among the "Unipolar" Extremity Leads, Circulation **5**:878, 1952.
6. Kesselman, R. H.: A New Synthesis of Some of the Physical Foundations of Clinical Electrocardiography, Am. Heart J. **47**:360, 1954.

7. Schwarzschild, M. M., Hoffman, I., and Kissin, M.: Errors in Unipolar Limb Leads Caused by Unbalanced Skin Resistances and a Device for Their Elimination, AM. HEART J. **48**:235, 1954.
8. Gentile, C.: La Resistencia Cutánea y su Importancia en Electrocardiografía, Rev. argent. cardiol. **22**:68, 1955.
9. Lepeschkin, E.: Artifacts in Multiple Channel Electrocardiographs Using a Common Central Terminal, AM. HEART J. **44**:62, 1952.
10. Lepeschkin, E.: Modern Electrocardiography. Vol. I, Baltimore, 1951, The Williams & Wilkins Company.
11. Goldberger, E.: Methods for Measuring the Resistance at Any Point on the Body and for Equalizing Inequalities of Skin Resistance, Brit. Heart J. **13**:549, 1951.
12. Jeanneret, P.: Clinical Measurement of the Electrical Resistance of a Patient With an Amplifier Type of Electrocardiograph, Brit. Heart J. **13**:43, 1951.
13. Edeiken, J.: Elevation of the RS-T Segment, Apparent or Real, in the Right Precordial Leads as a Probable Normal Variant, AM. HEART J. **48**:331, 1954.

MITRAL COMMISSUROTOMY IN THE YOUNGER AGE GROUP

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MITRAL commissurotomy is seldom performed in patients in the first and second decades of life. The natural history of rheumatic heart disease indicates that, at this age, the circulatory impairment, when present, is usually due more to an active pancarditis than to the mechanical obstruction at the mitral orifice. Futhermore, the operative stress, as we have learned from recent experience with commissurotomy in adults,[1,2] would make a reactivation of the rheumatic process more likely to occur.

However, we have recently observed several cases of mitral stenosis in very young patients whose markedly impaired cardiocirculatory condition was essentially due to the mechanical valvular obstruction, rather than to an active rheumatic process. It has been shown that children with mitral stenosis behave physiologically as adults with the lesion and, therefore, that the surgical relief of mitral valve obstruction can be beneficial at any age.[3]

The present report deals with results obtained in eleven patients between 8 and 16 years of age on whom mitral commissurotomy was performed. They are among 600 patients operated upon for mitral stenosis in our Cardiac Surgery Department.

ANALYSIS OF CASES

Status Prior to Admission (Table I).—Age of the patients ranged between 8 and 16 years. Six of them were boys and five were girls. A past history of rheumatic fever was present in five cases. Only one patient (Case 1) had suffered from a recent rheumatic attack. A history of tonsillitis or scarlet fever was present in the other cases. The diagnosis of rheumatic heart disease had been made in each case from one to seven years prior to admission. Exertional dyspnea, usually severe, was a common complaint. Paroxysmal nocturnal dyspnea and/or acute pulmonary edema had been present in four cases, hemoptysis in three cases. Right cardiac failure was present in the history of eight patients. All patients stated that their condition was gradually deteriorating prior to admission, in spite of cardiac therapy.

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TABLE I

CASE NO.	AGE	SEX	RHEUMATIC FEVER	HEMOPTYSIS	PULMONARY EDEMA	RIGHT HEART FAILURE	FUNCTIONAL IMPAIRMENT
1. M.G.	14	M.	+	−	−	+	Severe
2. L.G.	16	M.	−	−	−	+	Severe
3. S.F.	10	F.	−	−	−	+	Severe
4. D.T.U.	11	M.	−	−	−	−	Severe
5. B.I.	15	F.	−	+	+	+	Severe
6. I.N.	8	M.	+	−	−	+	Severe
7. A.G.P.	16	M.	−	+	+	+	Severe
8. M.C.	14	F.	+	−	−	−	Moderate
9. D.B.	12	F.	+	+	+	+	Severe
10. P.C.	15	F.	−	−	+	+	Severe
11. M.S.	13	M.	+	−	−	−	Severe

Findings on Admission (Table II).—Eight patients were in very poor general condition. Cyanosis was observed in four patients. In Case 7 it was so severe as to make us initially suspect the presence of a congenital cardiac malformation. All patients had regular sinus rhythm. The typical auscultatory findings of mitral stenosis were present in six cases; in two of them a mild aortic valve insufficiency was also suspected on the basis of auscultation. A harsh, mid-pitched Grade 3 to 4 systolic murmur was present over the cardiac apex in five cases in which the diastolic rumble of mitral stenosis was barely audible. Following a period of bed rest and digitalization, the loudness of the systolic murmur decreased and a diastolic rumble with an opening snap of the mitral valve was distinctly heard in each instance (Fig. 1). The heart was more or less enlarged in every case, but never as conspicuously as usually observed in active rheumatic pancarditis. The x-ray cardiac configuration did not differ significantly from the one usually seen in adults with mitral stenosis. Moderate left ventricular enlargement was present in five cases.

The electrocardiogram showed "mitral P waves" in ten instances and "pulmonary" P waves in one. P-R interval was normal in every case. Clear-cut electrocardiographic signs of right ventricular hypertrophy were present in seven patients. One patient had an incomplete right bundle branch block associated with right ventricular hypertrophy.

Clinical and radiological signs of pulmonary congestion were observed in all patients. Four subjects had hepatomegaly with tenderness in the right upper abdominal quadrant. There were no cases with ankle edema. Clinical or laboratory data suggestive of an active rheumatic process were absent. Angiocardiography was performed in five patients. The typical picture of mitral stenosis[4]

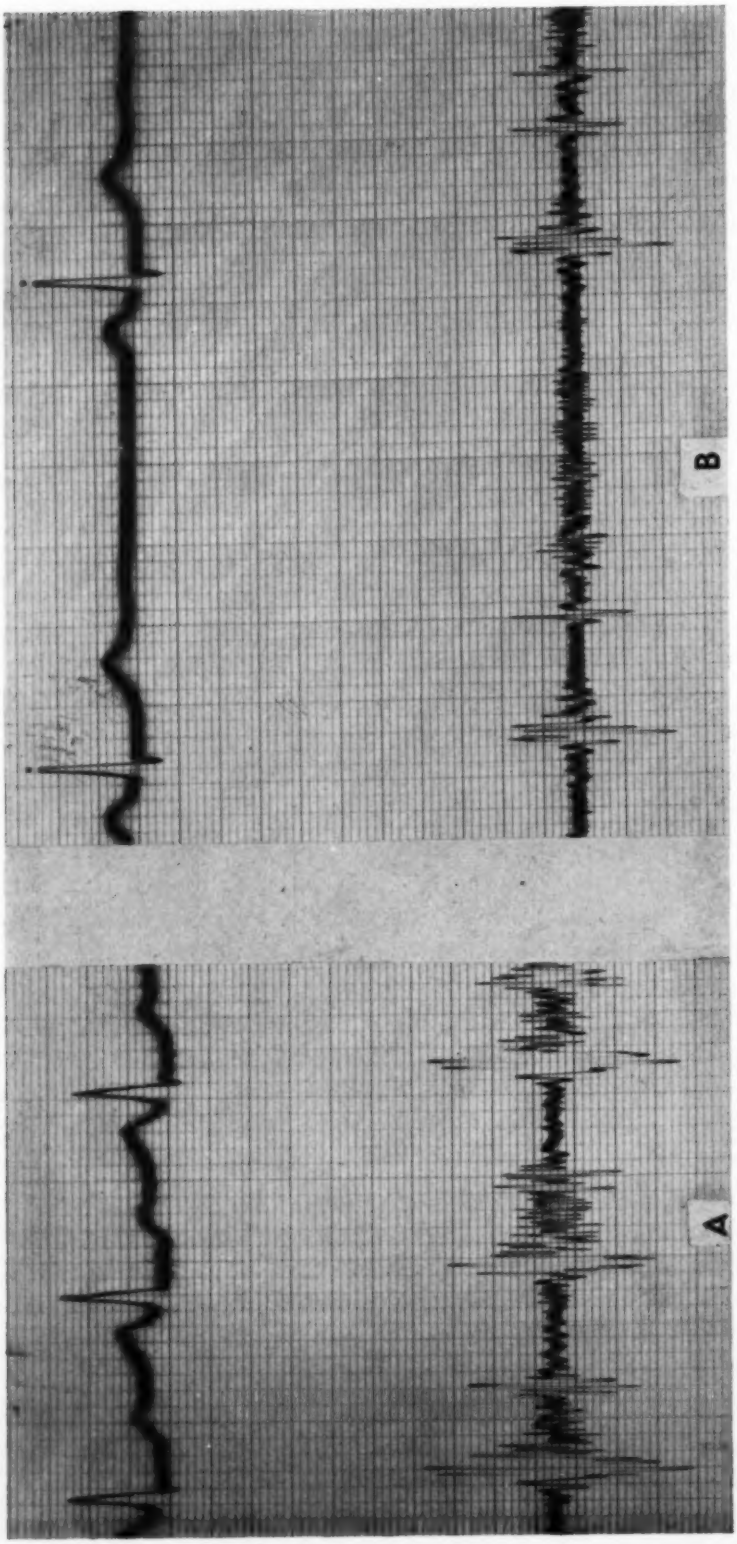


Fig. 1.—(Patient D. R., Case 9) *A*, Phonocardiogram recorded over the apex on admission (stethoscopic microphone, paper speed 50 mm. per second). Note the predominant systolic murmur. *B*, Apical phonocardiogram of the same patient taken after bed rest and digitalis therapy: the systolic murmur has almost disappeared and a diastolic rumble with an opening snap of the mitral valve is now clearly evident.

TABLE II

Functional Classification	Class I	0
	Class II	1
	Class III	5
	Class IV	5
Cyanosis		4
Regular sinus rhythm		11
Auscultation typical for mitral stenosis		6
Predominant apical systolic murmur		5
Cardiac enlargement (by x-ray)		11
a. Left atrium		11
b. Right ventricle, moderate		4
marked		7
c. Left ventricle, moderate		5
d. Pulmonary artery, moderate		6
marked		5
Pulmonary congestion		11
Hepatomegaly		4
ECG signs of right ventricular hypertrophy		7
Right ventricular hypertrophy and incomplete right bundle branch block		1
Mitral stenosis associated with mild mitral insufficiency		1
Mitral stenosis with mild aortic insufficiency		1

was present in each case. A severe degree of pulmonary hypertension was demonstrated in the two patients on whom cardiac catheterization was performed (Table III).

According to the Standard of the New York Heart Association, one patient belonged to functional Class II, five patients to Class III, and five patients to Class IV.

TABLE III. CATHETERIZATION FINDINGS IN CASES 5 AND 7

CASE	BEFORE COMMISSUROTOMY	AFTER COMMISSUROTOMY
7. A.G.P.		
Right ventricular pressure	110/30 mm. Hg	
Pulmonary artery pressure	110/80 mm. Hg	
Mean capillary pressure	30 mm. Hg	
Cardiac index	2.62 L.	
Total pulmonary resistances	2220 dynes/sec./cm.$^{-5}$	
Pulmonary vascular resistances	1180 dynes/sec./cm.$^{-5}$	
Mitral valve resistances	1040 dynes/sec./cm.$^{-5}$	
Peripheral resistances	2220 dynes/sec./cm.$^{-5}$	
Right ventricular work	3.83 Kg.M./min./sq.M.	
Mitral valve surface	1 cm.^{2}	
5. B.I.		
Right ventricular pressure	120/5 mm. Hg	80/5 mm. Hg
Pulmonary artery pressure	120/55 mm. Hg	80/40 mm. Hg
Mean capillary pressure	36 mm. Hg	24 mm. Hg
Cardiac index	1.47 L.	2.52 L.
Total pulmonary resistances	3000 dynes/sec./cm.$^{-5}$	740 dynes/sec./cm.$^{-5}$
Pulmonary vascular resistances	1500 dynes/sec./cm.$^{-5}$	255 dynes/sec./cm.$^{-5}$
Mitral valve resistances	1500 dynes/sec./cm.$^{-5}$	485 dynes/sec./cm.$^{-5}$
Peripheral resistances	3861 dynes/sec./cm.$^{-5}$	1345 dynes/sec./cm.$^{-5}$
Right ventricular work	1.28 Kg.M./min./sq.M.	2.01 Kg.M./min./sq.M
Mitral valve surface	0.5 cm.^{2}	2.5 cm.^{2}

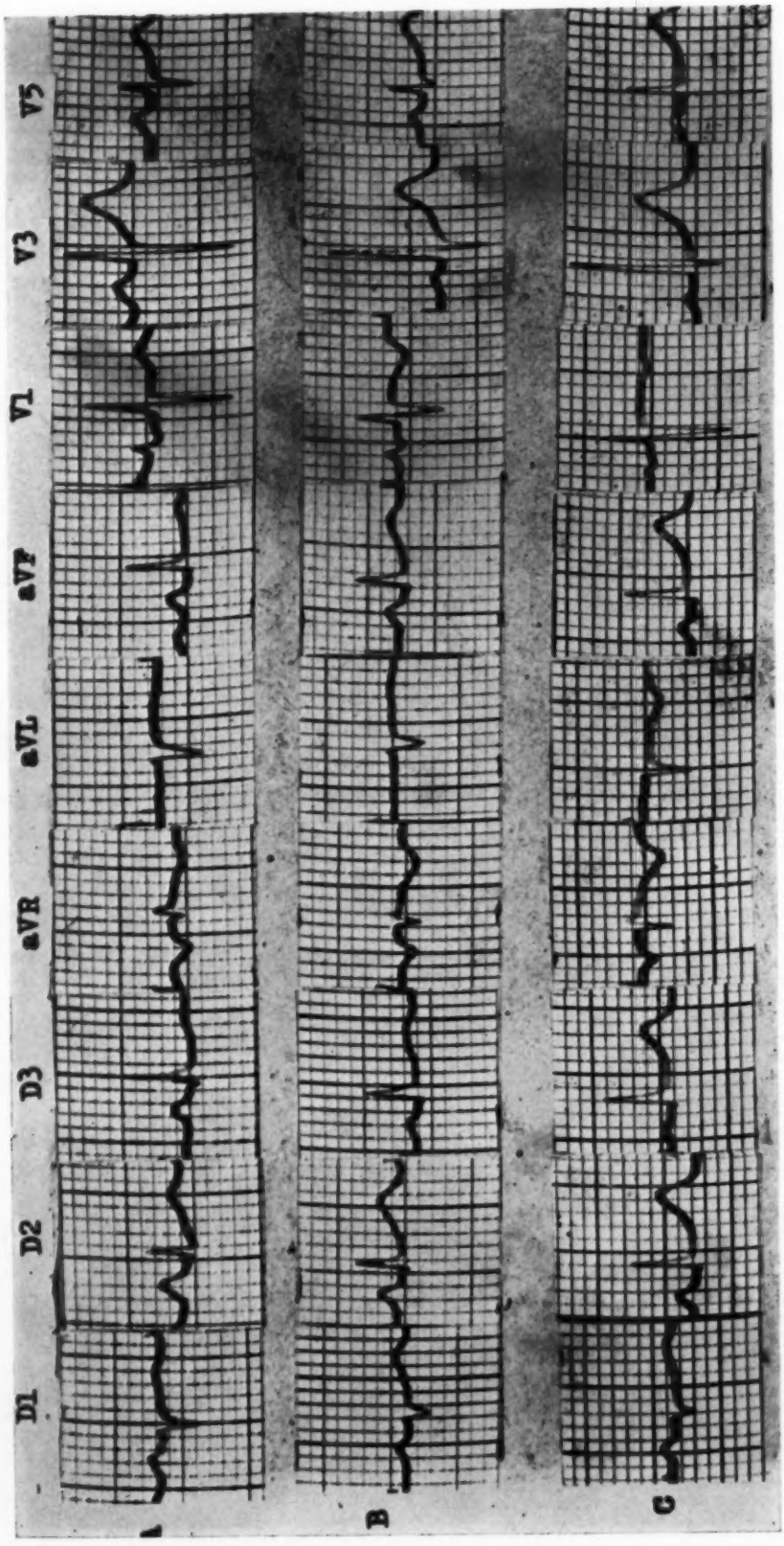


Fig. 2.—(Patient D. T. U., Case 4) Electrocardiogram taken before (*A*), three months (*B*), and twelve months (*C*) after commissurotomy. The precordial pattern shows a gradual evolution to normal.

Operative Findings.—A pure mitral stenosis was found in ten patients. In patient six there was an associated mild mitral insufficiency. The size of the mitral valvular orifice, as estimated by the surgeon according to a method previously described,[2*] was found to be quite small in every case but one, ranging between 0.2 and 1.2 cm.^{2} Only in Case 8 the mitral valvular surface was of about 2 cm.^{2} The valvular leaflets were particularly thickened in Cases 1, 2, 5, and 7. Digital divulsion was performed only twice. In the other cases it was necessary to perform a commissurotomy using Dogliotti's knife, ring.[2] In no instance was a surgically produced mitral insufficiency observed. The gross morphologic aspect of the left auricular appendage was not significant. Histologic studies were not carried out. In no case was there any evidence of thrombosis in the left auricle.

During the operative procedure significant cardiac arrhythmias did not occur in any case, except one in which a short run of ventricular tachycardia was recorded.

Immediate Postoperative Course.—Postoperative course was uneventful in eight patients. Fever lasted from eight to ten days with a gradual fall to normal. Mild signs of heart failure, rapidly controlled by the usual therapy, delevoped in Cases 1 and 8, in which a mild aortic insufficiency was also present. In Case 1 a rheumatic reactivation was suspected on the basis of fever, unexplained tachycardia, pallor, and joint pains about one week following commissurotomy.

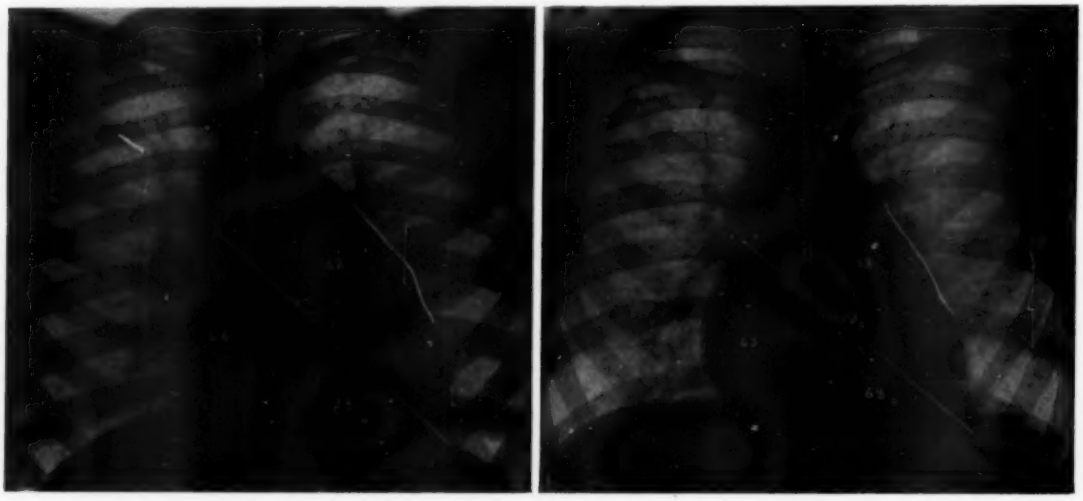
No deaths occurred in the group of cases. On the whole, the postoperative course of these children did not differ significantly from the one usually observed in adults with mitral stenosis.

Postoperative Follow-up.—Three patients were examined twenty months after commissurotomy, two after eleven to fourteen months, and the others after nine, four, four, and one months. Two patients are still under observation in the hospital. Results have been considered excellent in eight and good in three cases. Generally speaking, improvement was more marked in those patients whose follow-up period was longer. In these patients a noticeable weight gain and height increase had also occurred, well above the expected rate. In no case did hemoptysis and signs of left and/or right cardiac failure recur. Functional capacity was also greatly improved. Digitalis and mercurial diuretics were no longer necessary. Improvement was most dramatic in Case 9, whose mitral valvular surface was no more than 0.2 cm.^{2} The patient had been brought to the operating room in a desperate condition and the operation was carried out almost as an emergency procedure. At present she does not need any cardiac medication, she sleeps with one pillow, and twenty-five days after the operation, she could walk around in the Ward without trouble. The auscultatory findings did not change significantly. Improvement of the electrocardiographic pattern was observed at least three to four months following surgery (Fig. 2), and decrease

*A personal method of Professor A. M. Dogliotti for evaluating the degree of aperture of the mitral orifice consists in relating the operative finding to a chalk model of the index finger of the right hand.

in cardiac size was present in those patients who had undergone the operation at least one year previously (Fig. 3). Cardiac catheterization performed in Case 5, one month after the operation, confirmed the marked clinical improvement (Table III).

There were no recurrences of rheumatic fever during the period of observation. The presence of a mild aortic or mitral insufficiency did not seem to interfere with clinical improvement.



A. *B.*

Fig. 3.—(Same patient as in Fig. 2) Teleradiogram of the chest taken before (*A*), and one year after commissurotomy (*B*). Note the decrease of cardiac size in *B*.

DISCUSSION

Clinical, hemodynamic, and operative findings in our cases indicate that severe mitral stenosis can occur in the younger age group, perhaps more frequently than it is usually recognized. The size of the mitral valvular orifice in this series was below 1.2 cm.^{2} in all cases but one, and in Case 9 the degree of stenosis was so severe (0.2 cm.^{2}) that a very experienced surgeon found it quite difficult to localize the valvular opening. We also have recently observed a girl, age 11, admitted in severe cardiac failure, who died of acute pulmonary edema the day before surgery had been scheduled. The autopsy findings revealed that the valvular surface was no more than 0.5 cm.^{2} (Fig. 4). Marked anatomic changes of the pulmonary vessels were also present. On the contrary, there were no macroscopic nor microscopic signs of an active rheumatic process. When such situations exist, it is understandable how mitral commissurotomy could be a lifesaving procedure. It should also be pointed out that impairment of the lesser circulation can occur early in the course of rheumatic heart disease, and that commissurotomy can greatly improve functional pulmonary vascular changes. It is therefore conceivable that the appearance of anatomic changes can be delayed by the surgical procedure. Results of cardiac catheterization in Patients 7 and 5 demonstrate the seriousness of the impairment of the pulmonary vascular bed and the improvement which may follow the surgical relief of stenosis of the mitral valves.

Patient A.G.P. (Case 7), a boy aged 16, came under our observation with severe dyspnea, cyanosis, and signs of heart failure. The cardiac rhythm was regular and a harsh systolic murmur was heard at the heart apex. Angiocardiography showed stagnation of the contrast medium in the right side of the heart and in the pulmonary arteries. Systolic pressures of 110 mm. Hg were recorded in the right ventricle and in the pulmonary artery. Mean capillary pressure was about 30 mm. Total pulmonary resistances were 2.220 dynes/sec./cm.$^{-5}$, while pulmonary vascular resistances were higher than the resistances due to the stenosis itself. The work of the right ventricle was 3.83 Kg.M./min./sq.M. (Table III).

Patient B. I. (Case 5), a girl aged 15, was admitted in cardiac failure with a history of repeated hemoptysis and pulmonary edema on several occasions. In the right ventricle and in the pulmonary artery the systolic pressure was 120 mm. Hg. The mean capillary pressure was 36 mm. Hg.

Fig. 4.—Anatomic aspect of the stenosed mitral valves in a patient aged 9, who died of acute pulmonary edema. Note the extreme narrowing of the valvular orifice.

The total pulmonary resistances were 3,000 dynes/sec./cm.$^{-5}$ and the pulmonary vascular resistances were 1,500 dynes/sec./cm.$^{-5}$ The resistances due to the mitral valve were 1,500 dynes/sec./cm.$^{-5}$ The peripheral resistances were about 3,800 dynes/sec./cm.$^{-5}$ and, therefore, not significantly higher than total pulmonary resistances. Cardiac catheterization repeated thirty days after surgery showed a drop of the systolic pressure in the right ventricle and in the pulmonary artery, from 120 to 80 mm. Hg. Total pulmonary resistances decreased from 3,000 to 740 dynes/sec./cm.$^{-5}$ and pulmonary vascular resistances from 1,500 to 255 dynes/sec./cm.$^{-5}$ (Table III).

Reactivation of rheumatic fever in the immediate postoperative course does not seem to be more frequent in children than in adults operated upon for mitral stenosis. Furthermore, we have not observed any acute rheumatic process in our patients during the follow-up period. However, in view of the well-known high incidence of rheumatic reactivations in the younger patients, certain precautions seem necessary. Penicillin prophylaxis should be carried out

through the pre- and postoperative period according to the schedule which has been recommended for all young rheumatic patients. Administration of antirheumatic drugs as cortisone, ACTH, and salicylates for the prevention of rheumatic fever in patients undergoing mitral commissurotomy seems to be a useful procedure. Reports from authors of our group[6,7] indicated that several patients might benefit from this kind of treatment. Accordingly, cortisone, ACTH, or aspirin have been given to each of our patients, beginning three to four weeks before and continuing for a suitable length of time after the operation.

From our observations mitral commissurotomy would seem to be indicated in selected patients, even during childhood and adolescence. The conditions to be fulfilled are the presence of a serious cardiocirculatory impairment due to a severe, pure, or clearly predominant mitral stenosis and the absence of clinical signs of an active rheumatic process. The risk involved with commissurotomy in the younger patients is apparently no greater than in similar cases in the older age groups.

The results thus far obtained in patients with mitral stenosis in which commissurotomy has been performed during childhood and adolescence are most gratifying. Therefore, further observations on a larger number of patients seem justified.

SUMMARY

Out of 600 patients on whom mitral commissurotomy has been performed in the past four years, eleven ranged between 8 and 16 years of age.

All but one had a very tight mitral stenosis. Severe cardiocirculatory impairment was present in every case.

Results can be considered as very good or good in all patients. In no instance was reactivation of rheumatic fever observed in the follow-up period (from one to twenty months).

The presence of severe mitral stenosis and of marked cardiocirculatory impairment in absence of an active rheumatic infection is considered to be an indication for commissurotomy, even in patients in the younger age group.

Antirheumatic drugs (cortisone, ACTH, and aspirin) and antistreptococcal prophylaxis in the pre- and postoperative period seem to be a valuable adjunct to the surgical procedure.

SUMMARIO IN INTERLINGUA

Inter 600 patientes, in qui commissurotomia mitral esseva executate in le passate quatro annos, dece-un habeva etates de inter 8 e 16 annos.

Con un exception, omne le patientes del gruppo juvenil habeva un strictissime stenosis. Sever disturbationes cardiocirculatori esseva presente in omne iste casos.

Le resultatos obtenite in le juvene patientes pote esser considerate como bonissime o bon. Esseva observate nulle occurrentia de un reactivate febre rheumatic durante le periodos de observation postoperatori (de inter un e vinti menses).

Le presentia de sever stenosis mitral con marcate disturbationes cardiocirculatori e sin active infection rheumatic es considerate per nos como indication pro commissurotomia, mesmo in patientes de etates juvene.

Drogas antirheumatic (cortisona, ACTH, aspirina) e un prophylaxis antistreptococcal durante le periodo pre- e postoperatori es apparentemente de alte valor como adjunctos al procedimento chirurgic.

REFERENCES

1. Soloff, L. A., Zatuchni, J., Janton, O. H., O'Neil, T. J. E., and Glover, P. R.: Reactivation of Rheumatic Fever Following Mitral Commissurotomy, Circulation **8**:481, 1953.
2. Actis-Dato, A., and Angelino, P. F.: Commissurotomia per Stenosi Mitralica. Considerazioni clinico-chirurgiche su 200 casi e indicazioni operatorie, Cardiologia Pratica **5**:113, 1954.
3. Lurie, P. R., and Shumaker, H. B.: Mitral Commissurotomy in Childhood, Pediatrics **13**:454, 1954.
4. Actis-Dato, A., Angelino, P. F., and Zambelli, E.: L'angiocardiopneumografia nei vizi mitralici, Minerva med. **43**:693, 1952.
5. Massel, B. F.: Present Status of Penicillin Prophylaxis of Rheumatic Fever, Mod. Concepts Cardiovas. Dis. **20**:108, 1951.
6. Magri, G., Actis-Dato, A., Angelino, P. F., Jona, E., Levi, V., Molinatti, G. M., Pizzini, A., and Tedeschi, M.: Trattamento prolungato con ACTH negli operati di commissurotomia mitralica, Cardiologia Pratica **5**:203, 1954.
7. Dogliotti, G. C., Actis-Dato, A., and Angelino, P. F.: Primi risultati della terapia con ACTH in chirurgia cardiaca, Minerva pediat. **22**:1, 1952.

VASCULAR MODIFICATIONS INDUCED BY FLOW

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THE forms and the structures of the vascular system are generally attributed primarily to the influence of genetic forces and to their modifications by hormonal and pathologic processes.

Our recent studies on animals have suggested that significant elements of the structure of the blood vessels may result from the patterns of flow through them.[1-5] Thus, interference with the pattern of flow through a blood vessel may be followed by morphologic changes leading to a progressive narrowing of the lumen and to an ultimate structural reorganization of some of the elements of the vessel wall.[1-2]

This concept is supported herein by in vitro experiments in which changes in the "structure" of the walls of channels were produced by modifications of the patterns of flow in them. The results suggest that flow through a deformable channel can generate cushions and valvelike formations, produce stenotic "lesions," form excrescences and emboli, produce "dissection" of the wall, and determine other events bearing striking similitude to normal and pathologic structures seen in blood vessels.

METHODS

A reservoir maintained at a predetermined height provided a constant pressure head (Fig. 1). A tube of wide diameter permitted flow of water from the reservoir to two channels, 6 mm. in depth, cut into a plastic sheet. The channels used in the present study converged at an angle of 90 degrees. The opposing lateral margins of the channels were lined evenly with a layer of silicone putty.*

After applying a layer of silicone approximately 5 mm. in thickness to each side wall of the channel, a lumen of about 10 mm. in cross section remained.

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*Bouncing putty (SS 91 General Electric Company). This material was particularly suitable for the present study, since it has a specific gravity near that of water (1.1), it is water repellent, and it responds to continued pressure by deformation.

Care was taken to pack the silicone uniformly. A transparent plate was clamped firmly over the plastic sheet in which the channels were cut, permitting continuous observation of changes in the lining.

RESULTS

1. *Low Rates of Flow.*—In twenty experiments, water was permitted to flow continuously from the reservoir to the channels lined with the silicone. When the pressure head was 20 to 40 cm. water and the flow rate was 1.5 L. per minute or less, only slight changes in the structural pattern of the vessel walls were evident, even after two hours or more of flow. The rate of flow was practically unchanged during this time (Fig. 2).

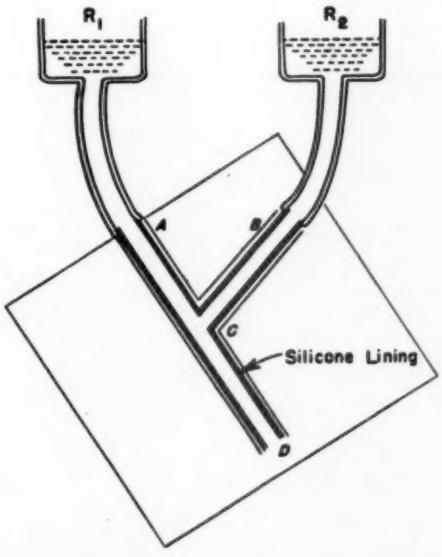

Fig. 1.—General design of the apparatus used. R_1 and R_2 represent water reservoirs maintained at fixed heights. In the present experiments these were combined into a single reservoir. Flow was divided into two streams, *A* and *B*, which joined at the confluence, *C*. Flow then continued through the continuing tube *CD*. The lumen of the channel, noted *ABCD*, was lined with silicone putty as indicated by the heavy black line. Described in text.

2. *Increased Rates of Flow.*—With higher pressure heads, the delivery and velocity obtained were increased at the onset of the experiments. Changes in the shape of the deformable material lining the channel were then seen (Fig. 3). For example, a pressure head in the range of 50 to 60 cm. of water produced rapid changes in the form of the walls, causing a narrowing of the lumen with a progressive reduction in the rate of flow.

3. *Cushion Formation.*—Twenty experiments with pressure heads of 50 cm. of water, or greater, resulted in a consistent heaping up of the silicone at a point immediately downstream to the convergence (Figs. 4 to 9), with the formation of cushions of this material on the opposing vessel walls. Occasionally the movement of the material toward the center of the stream was symmetrical; more commonly

it was asymmetrical. Sometimes formations began primarily on one wall, with the later appearance of a cushion on the contralateral side (Fig. 5).

4. *Progressive Stenosis.*—The cushions tended to enlarge progressively as flow continued, until they produced a relative stenosis, reducing the rate of flow despite an unchanged pressure head. Complete stenosis was seldom produced

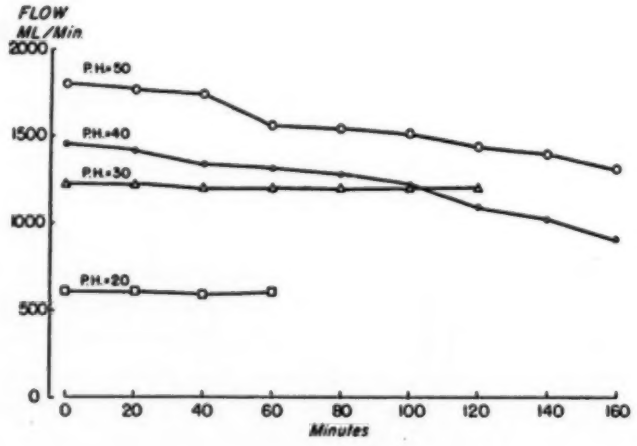

Fig. 2.—Rates of flow obtained at lower pressure heads. Delivery per minute is given in the vertical axis, and time of the experiment in minutes is given in the horizontal axis. The pressure heads (P. H.) used to obtain this flow is given in centimeters of water as numbers at the beginning of each line.

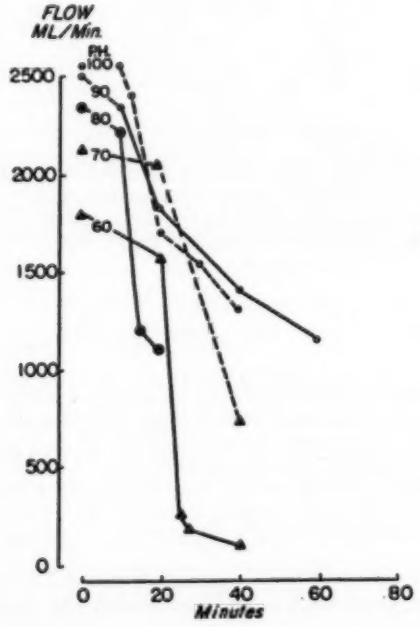

Fig. 3.—Rates of flow obtained at higher pressure heads. Conventions as in Fig. 2. The data of this figure differ from those in Fig. 2, in that with higher pressure heads, changes were produced in the lumen of the channels, and these had inhibiting effects on delivery.

since only a limited amount of silicone was available at any site, and the cushion therefore did not always extend throughout the entire depth of the channel (Fig. 9).

5. *Drag.*—After development of a cushion pair had taken place, the force of the flowing stream slowly pushed the wall of the cushion downstream, folding the cushions against the channel wall, usually with a reopening of the channel (Figs. 10 and 11). Other parts of the wall also moved slowly downstream, probably due to the drag induced by flow.

6. *Secondary Cushions.*—Cushions also formed at other sites downstream from the first formation (Figs. 6 to 8). After a single valve or cushion had been formed, the streamlines became disturbed, and cushionlike formations appeared at fairly regular intervals on the walls of the downstream channel, usually spaced about 5 cm. apart in the present model (Figs. 10 to 12).

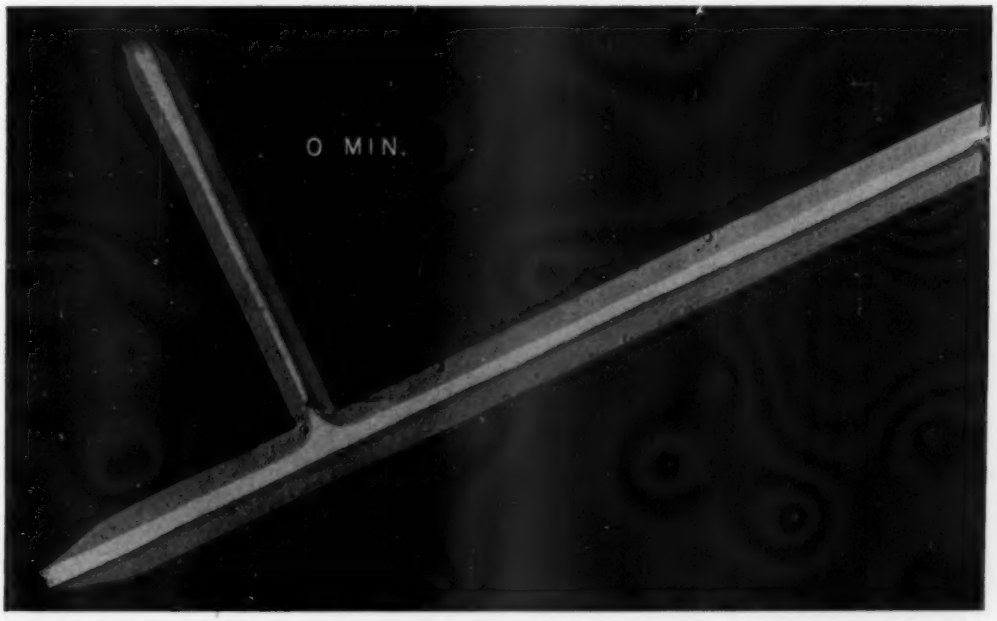

Fig. 4.—0 minutes. Flow has just begun. Air bubbles are seen leaving the stream at sites of increased pressure and of transmural flow from the lumen.

Figs. 4 to 12.—A series of successive photographs in a single experiment showing deformations of silicone putty through horizontally placed channels, as in Fig. 1. The view is through a transparent sheet of glass which forms the roof of the channel. The rigid walls consist of black plastic. The silicone putty, which has been packed as uniformly as possible against these rigid walls, is shown as the grey material. The bottom layer, forming the lower limit of the channel, is white. Nine photographs taken in succession are presented. A constant pressure of 45 cm. water supplied water to inlets at lower and upper left. The outlet was at the upper right. Air bubbles are seen to be trapped at various sites between silicone and the transparent plate.

7. *Inhibition.*—In some experiments cushions developed near the outfall (D) of the model at approximately the same time as other cushions were developing at the upstream site of convergence (Figs. 6 to 8). Whenever the cushions at the outfall developed rapidly, there was an inhibition of the rate of develop-

ment of the cushions further upstream (Figs. 6 and 7). After the outfall cushions had virtually closed and had then been washed away, the development of those at the convergence (C) began again and proceeded rapidly (Figs. 8 and 9).

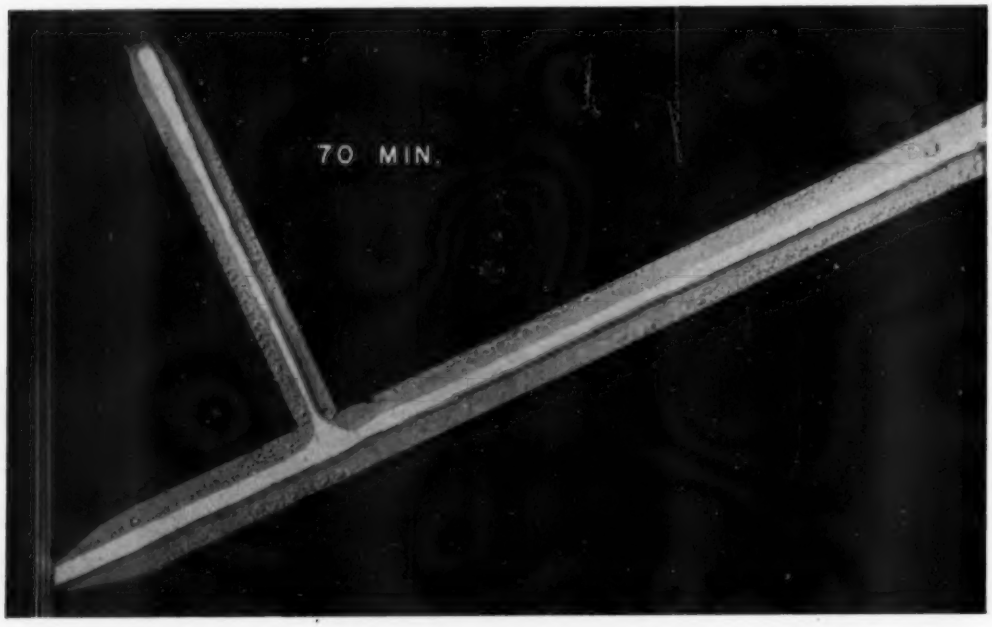

Fig. 5.—70 minutes. Cushions have formed just downstream to the confluence and at the outfall. A small dissection of the silicone from the rigid wall has appeared immediately below the confluence.

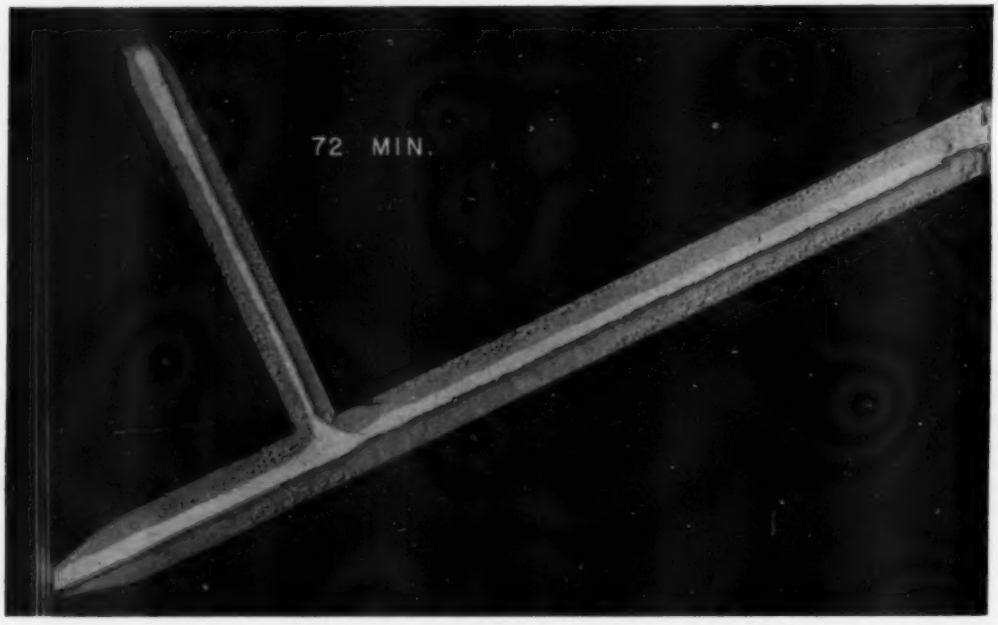

Fig. 6.—72 minutes. The cushions at the outfall have increased rapidly in size. A row of tiny bubbles has formed at the margin of the cushion at the confluence.

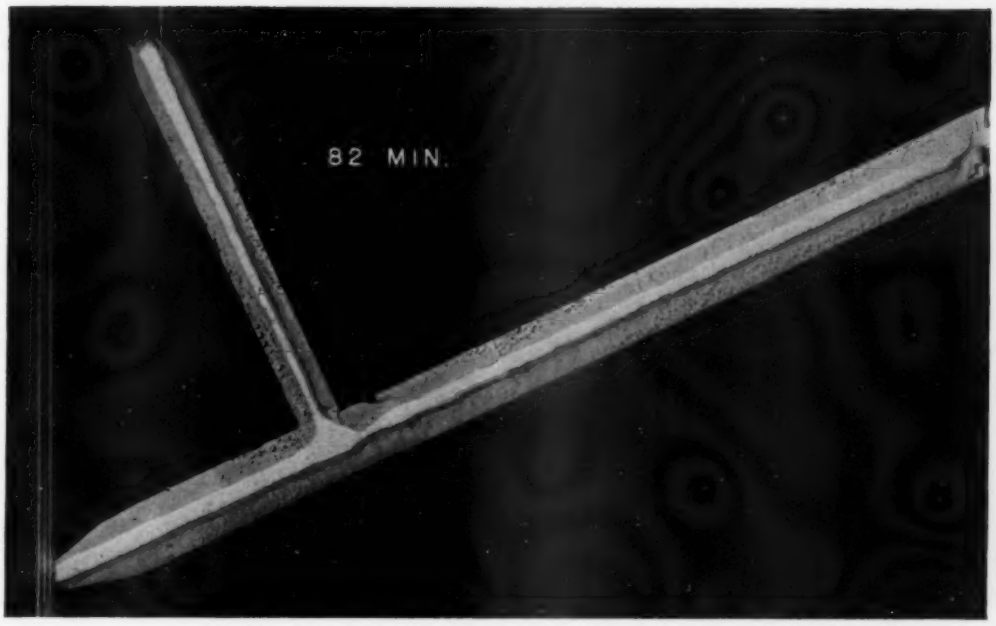

Fig. 7.—82 minutes. The outfall cushions have met and fused. Lines have formed in these cushions, the result of a transmural flow from the wall into the stream. The cushions at the confluence are enlarging.

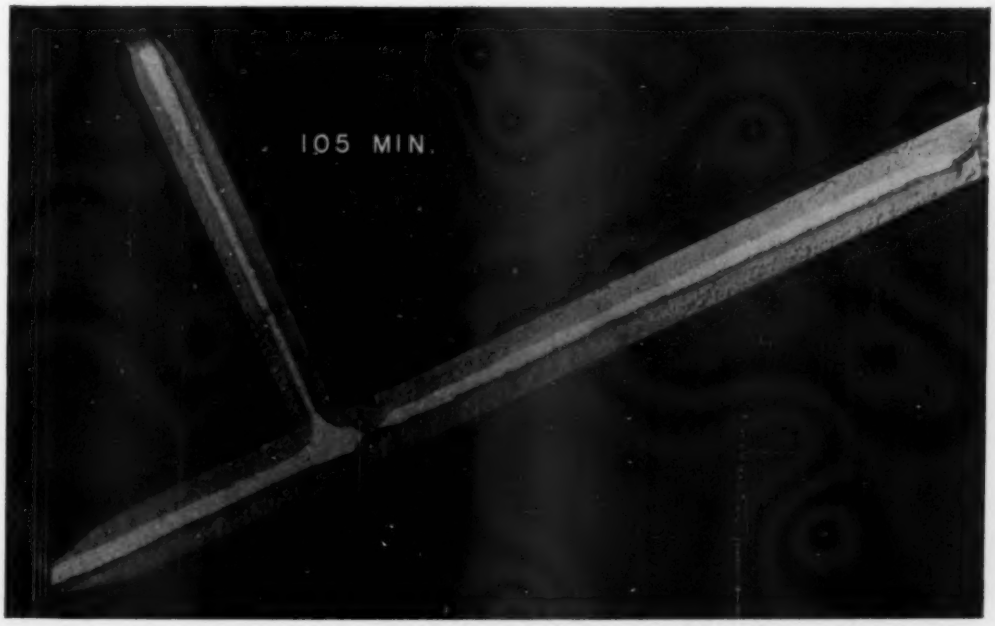

Fig. 8.—105 minutes. The outfall stenosis has been swept away, although this is not apparent from the view given. The cushions at the confluence have enlarged and have almost closed off the stream. The dissection has become larger. Irregularities have appeared just downstream to the stenosis. The increased number and size of bubbles upstream to the confluence indicate an enhanced transmural flow out of the stream.

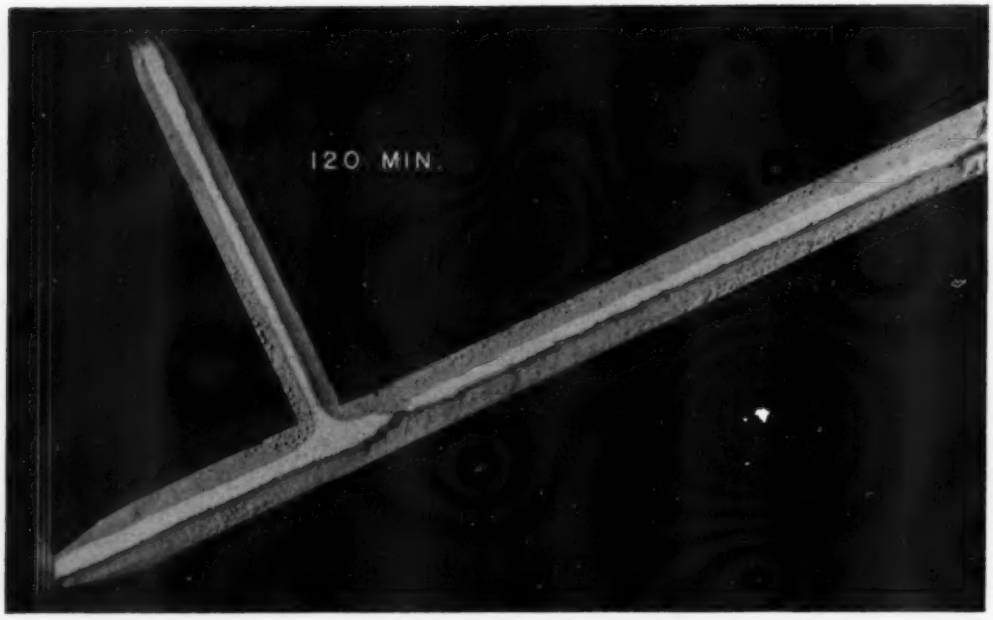

Fig. 9.—120 minutes. The stenotic site is being swept downstream with the formation of pseudovalves. (Formations bearing a very striking resemblance to true valves have been observed, but no example is seen in the present series.) Striations are present in the pseudovalves. The outlet obstruction has been washed away. The dissection is now receding.

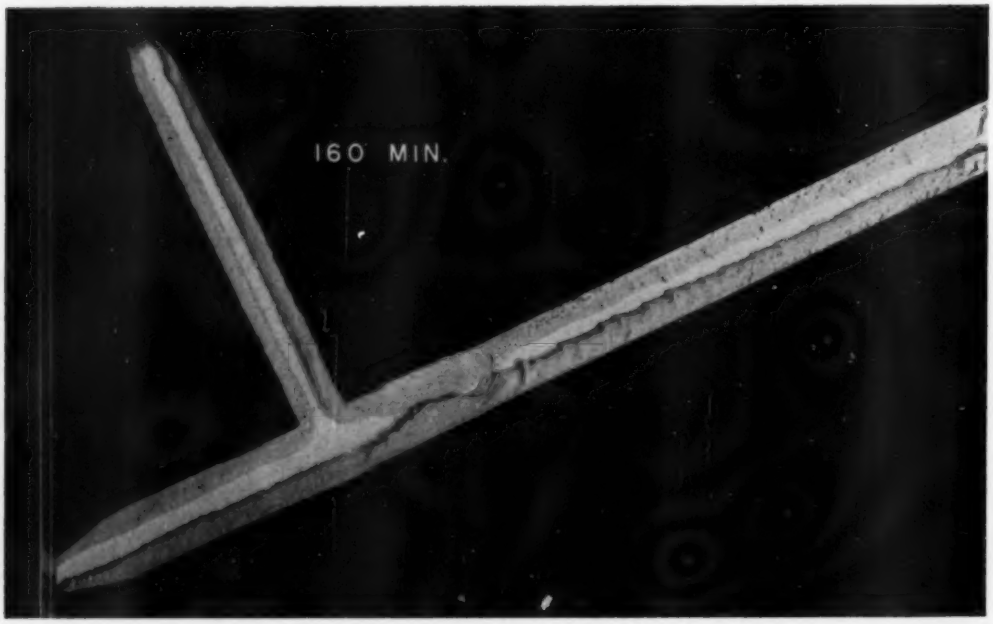

Fig. 10.—160 minutes. A long, narrow, stenotic channel has developed, showing a tendency to meander. A small cup-shaped structure (jet lesion) is present near the end of the meander. Other irregular formations have also appeared downstream to the primary disturbance. Note the relative lack of effect in the upstream portions of the channels.

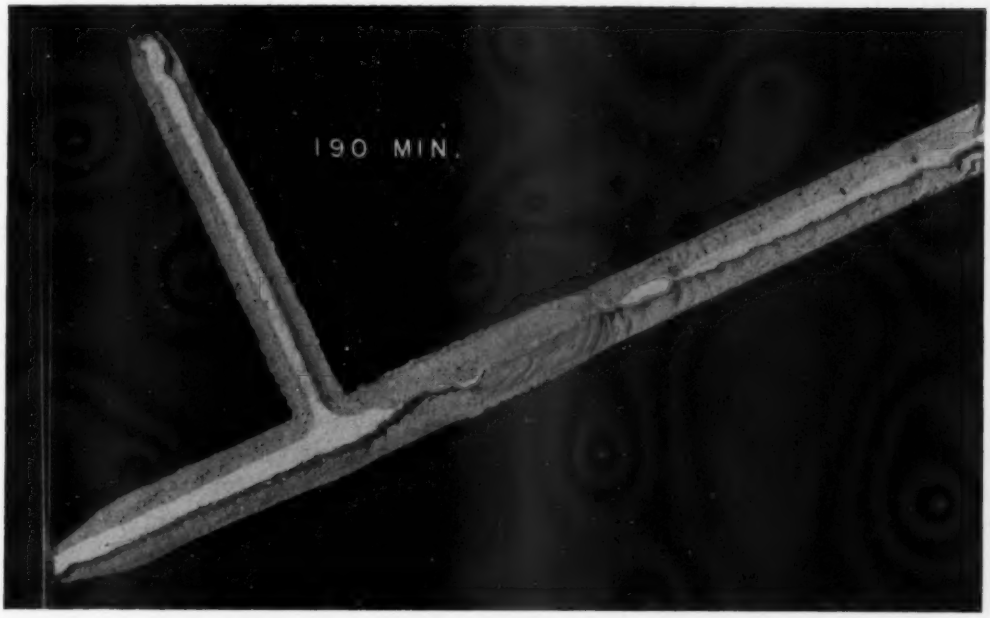

Fig. 11.—190 minutes. The meander process continues. Irregular formations appear. At sites of increased pressure small amounts of silicone have been forced between the black plastic wall and the upper transparent plate. This appears as an irregularity in the outer margin of the silicone. It occurs upstream to the stenosing cushions.

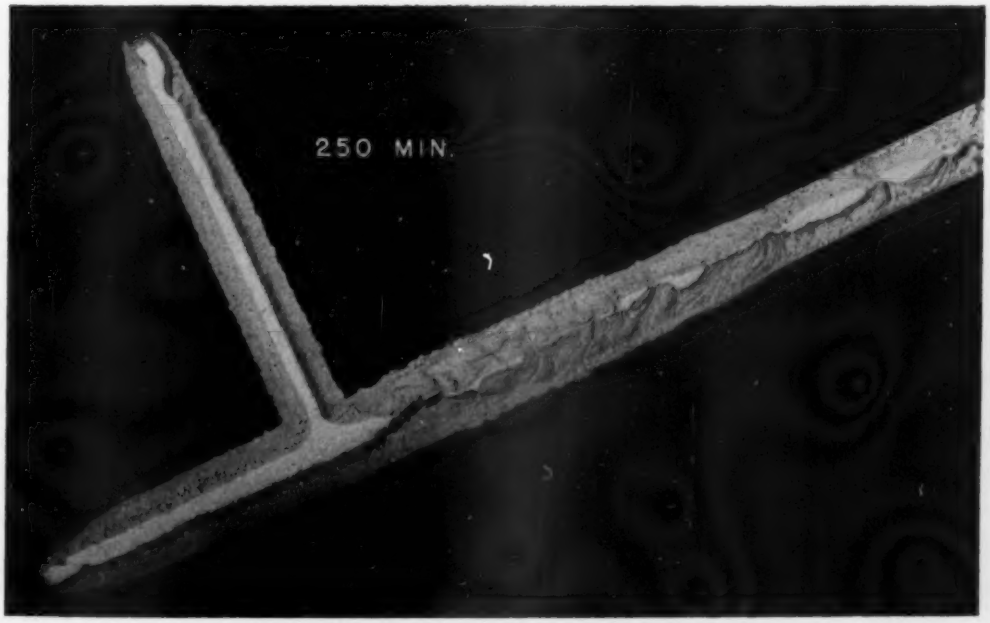

Fig. 12.—250 minutes. The meandering process continues. A large variety of formations appear. Silicone is being pushed against the upper plate near the first constriction, suggesting a spiral action of the stream at this site. Note that the upstream silicone has no distortions except at the inlet, where some disturbed flow patterns could not be avoided.

The development of cushions or valves upstream of the convergence (C) was rarely seen (Figs. 4 to 12).

8. *Valve Formation.*—In occasional experiments the downstream aspect of a cushion pair became rapidly hollowed out in such a way that only thin lips of silicone remained, with their free ends pointing downstream. The structures then had a superficial, but unmistakable resemblance to blood vessel valves.

9. *Vibrations.*—After the development of the valve pair, the "leaflets" were commonly seen to vibrate synchronously in the flowing stream. These would intermittently move toward each other and then move apart. The frequencies observed were of the order of 1 or 2 cycles per second.

10. *Pocketing.*—Immediately downstream from a site of orificelike narrowing, the deformable material was usually compressed with the resultant formation of a "pocket." This apparently resulted from the force of the jet acting on the wall (Figs. 10 and 11).

11. *Scour.*—At sites where the stenotic cushions had formed and the stream had practically closed off, the pressure on the upstream ends of the cushion caused them to be pushed progressively downstream, often folding the remaining cushion against the walls (Figs. 8 to 12). The orifice would then be widened progressively by the force of this stream as well as by a scouring action, with tags of the deformed cushions being dislodged and swept downstream.

12. *Streamers.*—Careful examination of the cushions and valvelike structures revealed that occasionally small tags or streamers (excrescences) of silicone extended from the trailing edge of the cushions or of the valvelike structures flapping in the stream (Figs. 10 to 12). The pedicle between the cushion (or valve) and the streamer then became more and more attenuated as it was stretched, and finally the tip of the streamer was pulled away and swept downstream as an "embolus."

13. *Lift.*—In some experiments the entire thickness of the deformable material lining the wall was suddenly lifted away from the rigid wall, forming a space between the outer matrix wall and the lining layer of the silicone (Figs. 5 to 8). This occurred commonly when the silicone was not packed solidly against the rigid matrix wall. However, on occasion, the silicone layer was lifted rather suddenly from the matrix (dissected) at points at which no apparent flaw in the application of the material was present.

When the lateral pressure acting on the wall was increased, as occurred when obstructions were produced downstream of the dissection, the dissection tended to disappear (Figs. 9 and 10).

14. *Effects on Flow.*—As the cushions developed and began to reduce the channel lumen, delivery through the tube decreased progressively. Typical results on the relation of cushion development and flow are given in Figs. 2 and 3.

While inspection of the channel from above sometimes gave the impression that the entire lumen was blocked, an orifice could usually be shown to remain at these sites. This limitation in closure resulted from the fact that the occlusion could be accomplished only by a movement of silicone from adjacent sites, and the

amount of material available in the model was necessarily limited. Further, various amounts of the material were dislodged by the scour process preventing the tendency to progressive obstruction.

15. *Meander.*—The development of a series of cushions, pocketing, and of scour action along the channel ultimately caused the stream to take on a meandering course (Figs. 10 to 12). In the present studies the meander pathway was ultimately limited by the rigid outer walls to which the deformable silicone had been affixed.

16. *Flow Mapping**.—An attempt was made to visualize the streamline patterns associated with the formation of the cushion by introducing dyes into the water passing through the channels, or by seeding the stream bed with crystals of potassium permanganate.[6] The streamlines were compressed immediately distal to the convergence, indicating higher velocities in these regions. Under some circumstances an intermittent fluttering of the line demarcating the two adjacent streams with the dye moving in and out at this point could be observed, pointing to an instability in the positions of the two merging streams.

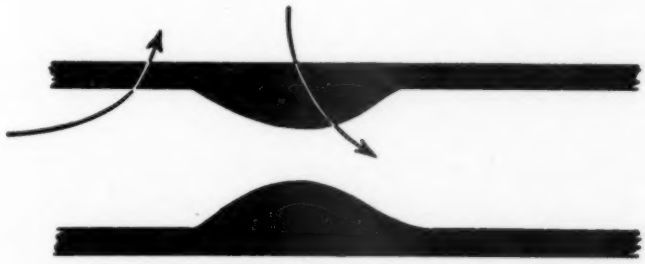

Fig. 13.—Schema illustrating transmural flow at a cushion. Flow is from left to right. The first arrow shows the usual direction of flow from the stream across the vessel wall (heavy black line) to the "tissue spaces." The second arrow, at the narrowing of the lumen between the cushions, shows the reverse pattern with flow from "tissue spaces" into the stream.

17. *Transmural Flow.*—In an attempt to obtain information concerning the pressures present at different sites along the channel, experiments were carried out in which the transparent upper plate was fitted relatively loosely to the plastic matrix. Under these circumstances, minimal amounts of water could move along the layer separating these two sheets.

Since small air bubbles were usually carried in the stream, the changes in the flow patterns taking place in the layer between the matrix and the transparent covering plate could be readily observed, permitting mapping of their routes (Figs. 5 to 12). At most sites, this flow was from the main stream across the silicone layer, escaping to the edges of the model. However, at those sites at which cushions were developing, a reversal of the usual pattern was seen. In

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*This set of experiments was carried out during the summers of 1953 and 1954, through the cooperation of Floyd Galler, B.S., who is at present a student of the Harvard University School of Medicine.

these instances, air bubbles which had passed from the main stream en route to the edge of the matrix gradually changed their direction and coursed back toward the stream, rejoining it at or slightly below the site of the narrowing. This behavior provided an appraisal of the pressure gradients in operation, this being from stream to wall at sites of relatively low velocities. At sites of high velocity, as occurs at narrowing of the stream, the low lateral pressure of the stream reversed the pressure gradient with a flow from wall, across silicone, and into the stream (Fig. 13).

The trajectory of the fluid and air bubbles returning to the stream ultimately produced groves which gave the appearance of small vessels coursing in the cushions or valves, and draining into the main stream (Fig. 9).

DISCUSSION

The foregoing experiments established a method for the study of the potential physical mechanisms which may be operative in valvular genesis and in stenotic closures of tube systems with walls composed of deformable materials.

Physical Considerations.—The induction and progression of cushions at particular sites, with the ultimate obstruction of the channels they line, can be shown to be related to the velocity of the stream at these points. These effects depend upon the principle of the conservation of energy.[7-9] Thus the lateral pressure must fall at a given point in a stream if the velocity increases at that site. Since the channels studied were lined with a deformable silicone putty, this pliable substance can gradually stream from sites of higher pressure to those of lower pressure. At a convergence where the velocity of the stream tends to be enhanced, the drop in the lateral or distending pressure will result in a tendency for a local heaping up of silicone into a hillock, or cushion (Fig. 14).

This effect is apparently inhibited when the lateral pressure remains high, such as occurs when a more distal resistance, as at the outlet, increases the distending pressure in the channel.[9] In the absence of such an inhibiting force, the process of narrowing tends to become progressive. This occurs because the cushion deviates the flow lines from their normal rectilinear path, futher accelerates the stream, and drops the pressure at this site; the conditions for progressive enlargement of the cushion are thereby produced and the process may continue until it leads to virtual obstruction of the stream.

Finally, when only a small opening remains, the resistance to flow increases rapidly in accord with Poiseuille's law, and the velocity of the stream at this point is reduced. The process of progressive narrowing thus comes to an end point with only a small orifice remaining. The heightened lateral pressure on the upstream walls of the cushion ultimately may force the accumulated material aside, reopening the channel. With increased flow, a sequence of new cushion formation and narrowings is then created at sites further downstream. The pocketing and scouring effects follow the changes in streamlines.

The production of valvelike structures appears to result from the fact that the high velocity flow through the narrowed orifice between the cushions produces such great momentum that the streamlines cannot follow the downstream form

of the cushion. Because of this, the streamlines "separate" from the wall, leaving a pocket of relatively static fluid.[10] In this region, the higher lateral pressure hollows out, or "pressure atrophies," the downstream end of the cushion, forming the valvelike structure.

The viscous forces of the fluid tend to drag the plastic material downstream. This effect is noted particularly at prominences such as occur at cushions and valves, although it probably also occurs, to a lesser extent, at all points along the stream. The high velocity of the stream impinging on some of the walls of the cushions, or on the valve tips, may detach small tags which form streamers. These may ultimately be detached, being swept downstream as "emboli."

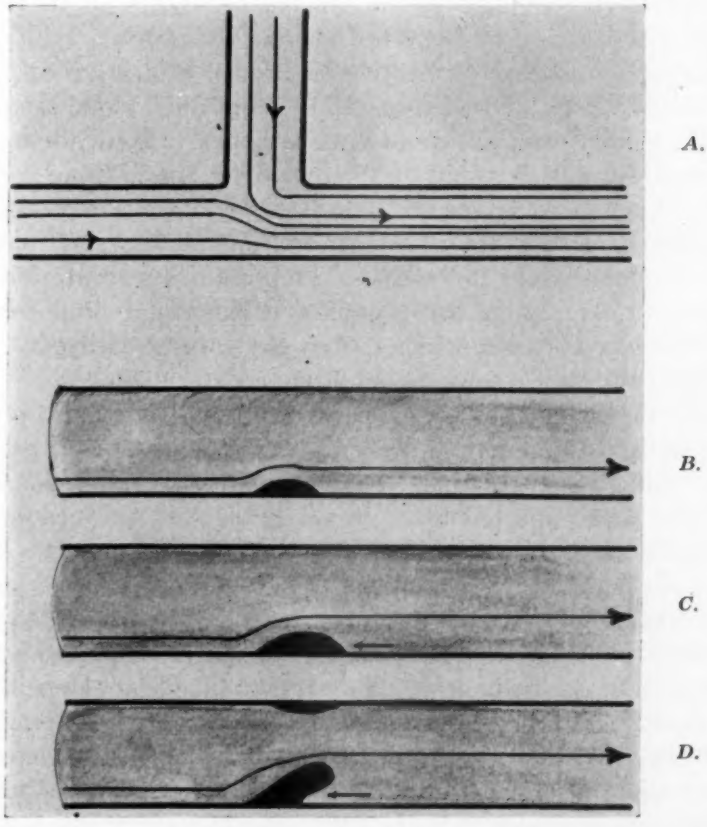

Fig. 14.—Schema, illustrating flow patterns leading to cushion and valve formation. In *A*, the streamlines impinge and cause acceleration of the stream, with a coincidental fall in lateral pressure at the convergence. *B* shows a streamline deviated and accelerated by a cushion, further dropping the lateral pressure at this site. In *C* this process continues and a region of separation, indicated by the short arrow, develops at the downstream end of the cushion. In *D*, the region of separation (and higher pressure) has caused "atrophy" of the downstream face of the cushion, producing a valvelike structure.

The lift phenomenon depends on the same dynamic factors. Thus, when the adherence of the silicone layer to the rigid wall is deficient, a high velocity, such as might occur at a cushion, can drop the lateral pressure on this pliable material, so that it is "lifted" from the matrix, dissecting it away from the wall. The space between silicone and rigid wall fills with ambient fluid under a relatively positive pressure, further increasing the tendency to dissection.

The transmural flow patterns illustrate that the usual course of the perfusing fluid is from the stream across the silicone wall to the surrounding spaces. However, the gradient may be reversed at narrowings of the stream, with flow taking place from the surroundings, across the vessel wall, back into the main stream.

Viewed from the point of view of blood vessel structure, the experiments with silicone provide certain interpretations of potential mechanisms of valvulogenesis, as well as of the progression of stenotic lesions, and associated effects.[4] While there can be no certain and direct translation of data obtained on models to living vessels, certain instructive similarities deserve attention.

Vascular Interpretations.—The intimal coat of blood vessels normally consists of a thin layer of flattened endothelial cells with small amounts of connective tissue separating it from the media. These intimal tissues have the potential, along with other connective tissues, of growing into and obstructing the lumen of the vessel. For example, if the contained blood is squeezed out of a segment of a blood vessel and both ends are then ligated, the growth potential becomes manifest and a rapid proliferation of the intimal layer completely occludes the vessel in the course of a few days.[11] Undoubtedly, there are inherent changes in the growth potential, depending on factors such as age, hormonal pattern, and inflammatory processes. The present interpretation holds that the available growth potential of the intimal layer is normally inhibited by the lateral pressure of the blood stream, which exercises a "pressure atrophy" on the intima, causing a flattening of the endothelial lining cells.

If, now, the lateral pressure is markedly diminished because of an enhanced velocity of the stream, as might occur at a confluence or at a partial narrowing, the local atrophic influence would be eliminated. The lining cells would no longer be compressed and inhibited from growth. Instead, they could round up and begin to proliferate, forming a hillock, or cushion.

With each increment of growth of the cushion, the narrowing of the stream results in a higher velocity and a still greater pressure drop. More growth of the intima could then take place and the lumen would be narrowed further. Progressive growth of the cushion would thereby be facilitated until the lumen was virtually closed. The present results suggest that stenotic tendencies may depend on the occurrence of critical velocities of flow through a vessel, or an orifice.

Examination of the nature of valvogenesis in veins, lymphatic vessels, and in the heart indicates that the formation of such valves follows patterns of flow similar, in striking respects, to those described for the silicone material we have used.[12-15] First, cushions develop bilaterally; then, at a certain time in development the downstream ends of the cushions become hollowed out and typical valves are formed.

In previous experiments with the production of fistulae between the pulmonary artery and the right atrium, we have noted that when such openings are small, they tend to close spontaneously over the course of time.[16] Similar closure occurs in other small defects produced artificially, as in ventricular septal defects and arteriovenous fistulae. It may be that forces produced by high velocity flow through such relatively small orifices may lead to progressive closure, as a result of the physical forces and the biological tendencies outlined above.

In such cases, the ultimate reduction and even elimination of a vascular communication may be expected if the velocity of flow through the channel is of sufficient magnitude. When the lumen of such a connection is relatively large, however, and a critical velocity of the stream is not achieved, closure may not be induced. In fact, the mechanical forces of stress and strain (hoop stress) would cause them to enlarge, as indicated in a separate study.[5] Such orientation and growth of cells subjected to physical strain have been demonstrated in tissue cultures.[17]

If the foregoing interpretation were correct, it would indicate that the formation of many of the valves of the cardiovascular system may derive from similar mechanical forces. Separation of flow at the downstream end of a cushion would result in an increase in pressure at this site, and local pressure atrophy would begin. A greater region of separation would be produced and the atrophying force would continue until the downstream end would be hollowed out, leaving a thin segment of tissue which, moving in the stream, could appose with its partner to prevent regurgitant flow.

Cushion and valve formations were inhibited by a resistance at the outlet.[9] This would conform with the fact that valves do not ordinarily form in arteries, where resistance to outflow from the arterial tree produces a relatively high lateral pressure throughout the arterial system.

Experiments with silicone demonstrate that small tags of material may be formed at the downstream ends of valves and cushions. These tags bear a striking similarity to the excrescences of Lambl, which are occasionally seen on valves, especially of the heart.[18] The possibility that such structures may result from the action of mechanical forces described above must be considered.

The tendency for the pedicles of the silicone tags to elongate and ultimately to break and be swept downstream suggests the possibility that this process may provide a source of embolization of the peripheral vascular bed. The process of scour also may operate to dislodge elements of a stenosed region, and produce similar embolic effects. Should such tags of intimal tissue be dislodged and swept downstream, they would occlude peripheral arterioles. The fact that these are true emboli might not be immediately evident on pathologic examination, since the embolic tissues would consist of normal elements of the vessel wall and, thus, might be misinterpreted as a local (in situ) proliferation.

The development of the sinuous course of the meandering stream[19] has certain similarities in form to the tortuous course of many veins and sclerotic arteries. It is possible that hydraulic forces similar to those observed in these model experiments may play a role in the development of such an elongation and tortuosity of a vessel. Static factors may also contribute to elongation and tortuosity, as illustrated in a separate study.[5]

The lift produced by high velocity flow in the silicone model provides an approach to potential mechanisms leading to "dissections" such as occur in the aorta, and to subintimal hemorrhage in other arteries.[20] One of the unexplained paradoxes in dissecting aneurysms has been the occurrence of subintimal bleeding from small vessels. These hemorrhages take place despite the fact that these small vessels have relatively low blood pressures compared to the much higher compress-

ing force present in the aortic chamber. It would be expected, on first inspection of the problem, that the higher aortic pressure should act as a "compression bandage" to prevent subintimal hemorrhage and dissection, but this apparently does not happen. The present results would interpret the chain of events of dissection as due to a drop in lateral pressure at a site of high velocity. Such an effect might occur at a raised arteriosclerotic plaque which protrudes into the lumen, deviating and accelerating the adjacent streamlines. This drop in lateral pressure at such a site may be sufficient to produce a relative "suction" and lift the intimal plaque from the medial layer of the vessel wall, tearing its connection with the media and permitting subintimal hemorrhage and dissection. Dissection would, of course, be facilitated if the cementing substances of the media were deficient, such as occurs in Erdheim's cystic necrosis. An operational approach of this type may clarify the potential physical relationships in these important conditions, and provide a basis for further experimental work.

The directions of transmural flow of fluid at various sites also furnish suggestions concerning potential nutritive patterns acting on the vessel wall (Fig. 13). At sites of high positive lateral pressure, plasma containing oxygen and nutrients may be expected to filter continuously from the blood plasma, across the thickness of the vessel wall. Such filtered fluids might ultimately be collected in the subjacent vasa vasorum and lymphatics, and be carried to the venous return. However, at sites of high velocity produced at cushions and at thickened (pathologic) valves, reversal of the pressure gradient could cause tissue fluids to pass from the medial layers through the intima, returning thence to the blood stream. Under such circumstances, the intima might not receive the high oxygen content and the nutritive materials normally available to it, leading to structural changes and perhaps producing a site of reduced resistance to endovasculitis.

CONCLUSIONS

The present studies indicate how mechanical forces can affect the structure of a deformable channel, leading to various modifications, including stenoses, valve production, excresences, and other changes.

In studies to be reported later, the use of other matrices, such as semicircular channels, has been shown to develop "segmental arterial outlets" and "spiral valves" which also bear semblances to structures present in the cardiovascular system.

It is appreciated that these represent only in vitro situations. However, the striking similarities to processes found in the cardiovascular system suggest the possibility that mechanical factors may be responsible for significant elements of the development and structure of the cardiovascular system.[17-19] Further exploration of the potential influence of these mechanical factors, using plastic models of the general types described, can provide a basis for an operational approach to problems of blood vessel structure and disease.*

---

*L. Aschoff long ago suggested that thromboses were dependent on forces similar to those which cause the building of sand banks in rivers (thrombose und Sandbankbildung, Beitrage zur pathologischen anatomie und pathologie, **52**:2-5-212, 1912).

SUMMARY

1. Flow at critical velocities produced changes in the form of channels lined with a deformable silicone, with the development of structures superficially resembling those seen in blood vessels. These effects are attributed to the operation of mechanical forces.

2. At sites of high velocity, such as occur at confluences or at large pressure gradients, the silicone tended to form bilaterally symmetrical cushions. These increased in size until they virtually occluded the lumen, acting as a stenosis and reducing the flow delivery through the system. In some of these cushions, the downstream end was suddenly hollowed out, producing a valve-like structure.

3. At sites of high stream velocity, as at cushions, the silicone was sometimes pulled away from the rigid outer wall of the channel, producing a "dissection" of the wall.

4. The downstream ends of the cushions, or valves, commonly developed polypoid "streamers" whose peduncles became progressively elongated and attenuated; these masses finally broke free, being swept downstream as "emboli."

5. Transmural perfusion was normally from stream to wall. At sites of cushion formation, stenosis, or valve formation, the pattern was reversed, with flow taking place from wall to stream.

6. Similitudes of these formations and processes to those occurring in the cardiovascular system are discussed.

SUMMARIO IN INTERLINGUA

Le examine de materiales archival de catheterisation cardiac revelava un criterio pro le differentiation de duo distincte typos de stenose pulmonic infundibular. Isto es possibilemente importante in le determination del curso chirurgic in casos de iste disordines. Quando le crestas del pulsos de pression in le infundibulo e in le ventriculo dextere esseva synchrone, iste facto suggereva que un relativemente fixe (fibrose) orificio limitava le fluxo in avante verso le arteria pulmonar. Tamen, in le majoritate del casos examinate, le crestas in le duo cameras non esseva synchrone: le pression infundibular attingeva su cresta tosto in le systole e postea comenciava abassar se, ben que le pression ventricular continuava accrescer. Isto pote possibilemente explicar se per le contraction de un banda muscular al infundibulo. In un caso de iste typo, un "tunnel" muscular esseva trovate al operation. Un tal variabile stenose poterea esser partialmente responsabile pro un derviation bidirectional durante le systole.

The excellent technical assistance of Mr. Matthew Thompson is gratefully acknowledged.

REFERENCES

1. Rodbard, S.: Experimental Production of Valve-like Structures in Blood Vessels by Modification of Streamlines of Flow, Proc. Int. Physiol. Cong. **19**:710, 1953.
2. Rodbard, S.: Molding of Blood Vessels, Valves and Stenotic Lesions by Hydraulic Forces, Proc. Second World Congress of Cardiology **2**:50, 1954.
3. Rodbard, S., and Williams, C. Bolene: Cholesterol and Vascular Responses to Induction of Coarctation of the Aorta in Chicks, Circulation **8**:442-443, 1953.

4. Rodbard, S.: Factors in the Progression of Stenotic Lesions, In preparation.
5. Rodbard, S., Teitelman, S. L., and Zimmerman, L. M.: Mechanical Factors in Vascular Dilatations, Angiology, In press.
6. Moore, A. D.: Fields From Fluid Mappers, J. Appl. Physics **20**:790-804, 1949.
7. Bernoulli, D.: Hydrodynamica, Sive, de Viribus et Motivibus Fluidorum Commentarii, Argentorati, 1738, J. R. Dulsecker.
8. Rodbard, S., and Saiki, H.: Flow Through Collapsible Tubes, AM. HEART J. **46**:715-725, 1953.
9. Rodbard, S.: Flow Through Collapsible Tubes: Augmented Flow Produced by Resistance at the Outlet, Circulation **11**:280-287, 1955.
10. Goldstein, M.: Modern Developments in Fluid Dynamics, Oxford, 1938, Clarendon Press.
11. Geiringer, E.: Intimal Vascularization and Atherosclerosis, J. Path. & Bact. **68**:201-211, 1951.
12. Kampmeier, O. F., and Birch, C. L. F.: The Origin and Development of the Venous Valves, Am. J. Anat. **38**:451-499, 1927.
13. Bremer, J. L.: An Interpretation of the Development of the Heart, Am. J. Anat. **42**:307-337, 1928.
14. Chun Chang: On the Reaction of the Endocardium to the Bloodstream in the Embryonic Heart, With Special Reference to the Endocardial Thickening in the Atrioventricular Canal and the Bulbus Cordis, Anat. Rec. **51**:253-265, 1932.
15. Patten, B. M., Kramer, T. C., and Barry, A.: Valvular Action in the Embryonic Chick Heart by Localized Apposition of Endocardial Cushions, Am. J. Anat. **102**:299-312, 1948.
16. Rodbard, S., and Wagner, D.: Bypassing the Right Ventricle, Proc. Soc. Exper. Biol. & Med. **71**:69-70, 1949.
17. Bourne, G. H., Editor: Cytology and Cell Physiology, ed. 2, New York and London, 1951, Oxford University Press.
18. Raeburn, C.: Papillary Fibro-elastic Hamartomas of the Heart Valves, J. Path. & Bact. **65**:371-373, 1953.
19. Straub, L. G.: Mechanics of Rivers, Chap. XIIIC, pp. 614-616 of Hydrology, N. Y., 1949, Dover Pub.
20. Drury, R. A. B.: The Role of Intimal Hemorrhage in Coronary Occlusion. J. Path. & Bact. **67**:207-215. 1954.

Clinical Report

WOLFF-PARKINSON-WHITE SYNDROME OCCURRING IN BROTHERS

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THE occurrence of Wolff-Parkinson-White syndrome in siblings has only occasionally been reported. Öhnell[1] tells of one family in which four of six siblings were afflicted and another family in which five of ten siblings had paroxysms of tachycardia. Of the five subject to paroxysmal tachycardias, two had Wolff-Parkinson-White syndrome. Willis and Shepard[2] tell of a family in which three of six siblings were affected. Recently the father of these children was found to have evidence of Wolff-Parkinson-White syndrome.[3] The occasional appearance of this syndrome in infants[4,5] lends further support to the thesis that a congenital abnormality may be responsible for the ECG findings.

This report concerns two brothers who have recently been found to have short P-R intervals and long QRS durations. These two brothers have an older brother and a younger sister, both of whom are well and free of episodes of tachycardia. The father died in 1937 at the age of 74, probably from a coronary occlusion; the mother is 79 years old, and apparently free of heart disease. Electrocardiograms show no conduction abnormalities in the parents or siblings.

CASE REPORTS

CASE 1.—G.B. is a 57-year-old man, who, in his lifetime, has had five episodes of paroxysmal tachycardia. The first two occurred at the ages of 10 and 12, the last three in 1951, 1952, and 1954. There have been no other cardiac symptoms. The blood pressure is 134/90 mm. Hg. The heart sounds are normal and no murmurs are heard. By fluoroscopy the heart is normal in size. His electrocardiogram is reproduced in Fig. 1.

CASE 2.—W.B. is a 50-year-old man, and younger brother of G.B. He has had considerable cardiac anxiety for many years, based largely on premature beats and the knowledge that his brother had "heart trouble." In October, 1954, he had his first attack of paroxysmal tachycardia. This attack came on in the middle of the night, awakening him from a sound sleep, lasted 5 to 10 minutes, and then abruptly stopped. In the next three months he had three more such nocturnal attacks of palpitation. He is very active physically and, apparently, entirely free from anginal

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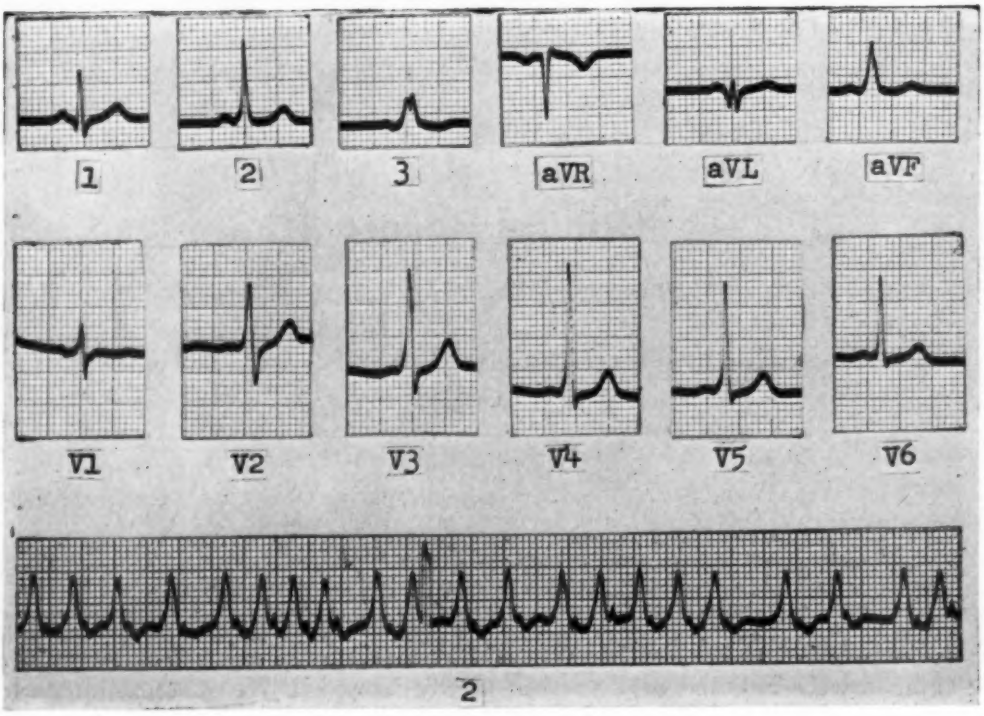

Fig. 1.—Electrocardiogram of G.B. The strip of Lead II at the bottom was taken during an episode of tachycardia in 1952.

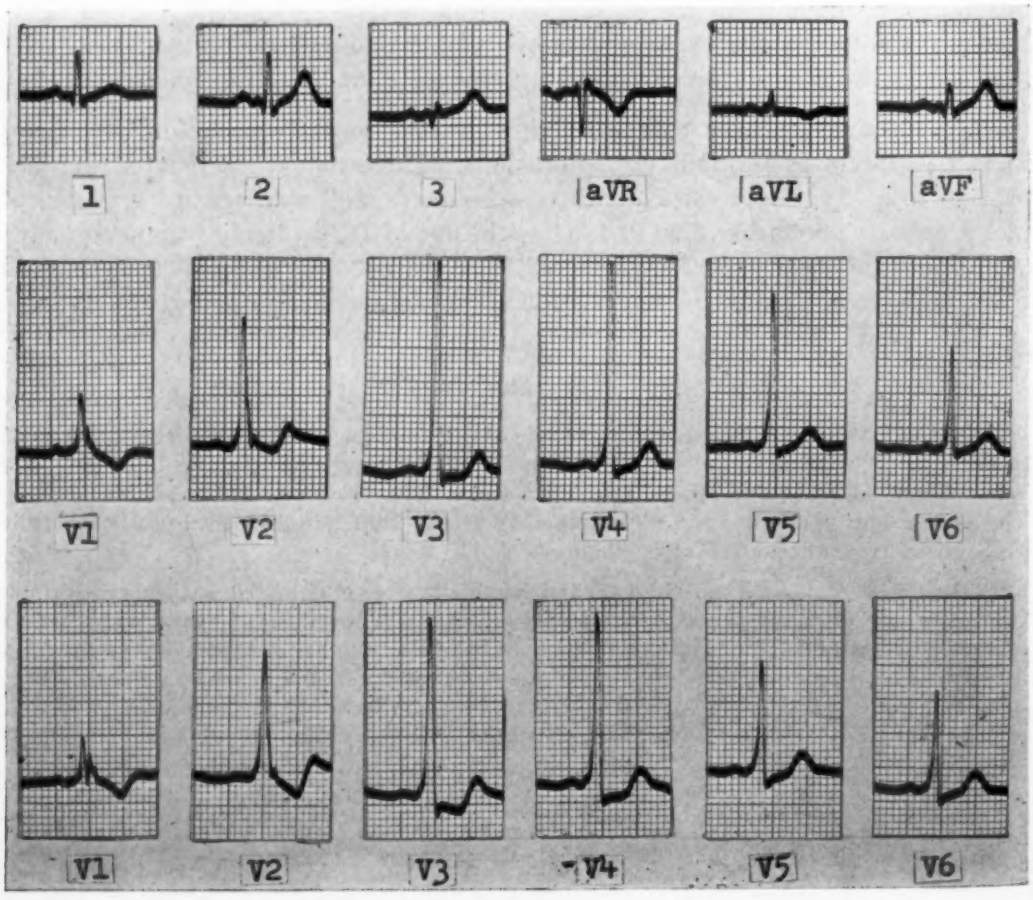

Fig. 2.—Electrocardiogram of W.B. *A*, Taken while at rest; *B*, Taken directly after exercise.

symptoms. The blood pressure is 130/80 mm. Hg. The first sound at the cardiac apex is reduplicated. All the heart sounds are of normal intensity and no murmurs are present. A chest x-ray film shows the cardiac silhouette to be normal in size with a cardiothoracic ratio of 14.7/31.0 cm.

His electrocardiogram (see Fig. 2) shows a short P-R interval and a prolonged QRS duration. Atropine, 1.0 mg. subcutaneously, and exercise do not alter the A-V conduction. Exercise (50 trips over steps used in doing the "2-step" exercise test) does not produce untoward symptoms, but does produce S-T depressions, particularly in the precordial leads. The exact significance of this is not clear; it is probably a physiologic reaction. The precordial leads are somewhat unusual, in that there is delay of the intrinsicoid deflection in V_6 and V_1, as well. A similar tracing is reproduced in a report by Boyer.[6]

SUMMARY

Two cases of Wolff-Parkinson-White syndrome occurring in middle-aged healthy brothers, aged 50 and 57 years, are reported. This rare occurrence, also reported by others, suggests that heredity may, at times, play a role in the appearance of this syndrome.

SUMMARIO IN INTERLINGUA

Es reportate duo casos del syndrome de Wolff-Parkinson-White, occurrente in fratres valide de 50 e 57 annos de etate. Iste rar phenomeno, etiam reportate per altere autores, suggere que factores de hereditate es a vices involvite in le occurrentia de iste syndrome.

REFERENCES

1. Öhnell, R. F.: Pre-Excitation a Cardiac Abnormality, Stockholm, 1944, P. E. Norstedt & Söner.
2. Willis, W. H., and Shepard, C. C.: The Familial Incidence of Certain Unusual Diseases, North. New York M. J. **10**:19-23, 1953.
3. Willis, W. H.: Personal communication.
4. Paul, O., and Harrison, C. J.: Wolff-Parkinson-White Syndrome in an Infant, J.A.M.A. **149**:363-364, 1952.
5. Gleckler, W. J., and Lay, J. V. M.: Wolff-Parkinson-White Syndrome and Paroxysmal Tachycardia in Infancy, J.A.M.A. **150**:683-685, 1952.
6. Boyer, N. H.: The Wolff-Parkinson-White Syndrome, New England J. Med. **234**:111-114, 1946.

Announcements

THE AMERICAN COLLEGE OF CARDIOLOGY has inaugurated a CLINICAL WORKSHOP PROGRAM, which furnishes person to person contact in the office, clinic, hospital, and in the laboratory with the most outstanding living pioneers in scientific and clinical cardiology. Gabriel F. Greco, M.D., Ozone Park, N. Y., Chairman Publicity Committee, Clinical Workshop Program, has announced that the program is available free to all members of the medical profession. Applications will be honored in order of their arrival. Workshop centers have been made available in North and South America, Canada, Argentina, and Mexico. Among the more than seventy preceptors are: James Watts, Director, National Cardiovascular Division, National Heart Institute, Bethesda, Md.; Ignacio Chavez, Director, Institute of Cardiology, Mexico; Hans Selye, Director of the Institute of Medicine and Experimental Surgery, University of Montreal; Dr. Pedro Cossio, Professor of Cardiology, Buenos Aires, Argentina; Dr. George W. Calver, Attending Physician, Congress of the United States, Washington, D. C.; Dr. Samuel A. Levine, Professor of Medicine, Harvard University, Boston, Mass. Application blanks may be obtained from Dr. Joseph B. Wolffe, Chairman, Clinical Workshop Committee, Valley Forge Heart Institute, Fairview Village, Pa.

THE FOURTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST OF THE AMERICAN COLLEGE OF CHEST PHYSICIANS will be held in Cologne, Germany, from Aug. 19 to 23, 1956, under the patronage of the Federal Chancellor Dr. Konrad Adenauer. The first Congress after the war was held in Rome, in 1950, the next one in Rio de Janeiro, in 1952, and the third one in Barcelona, in 1954. Eighty-six countries will send their representatives.

Prof. Dr. med. Dr.h.c. Gerhard Domagk will be the President, Prof. Dr. med. Dr.h.c. H. W. Knipping the Vice-President, Prof. Dr. med. J. Jacobi the General Secretary, and Prof. Dr. med. J. Hein the Chairman of the Executive-Committee of the Fourth International Congress.

The main subjects which will be discussed at the Congress deal with the problems of coronary diseases (diagnosis, pathophysiology, and surgery), industrial diseases of the chest, tuberculosis, lung and heart function, and tumors of the mediastinum. Several outstanding foreign and German scientists and clinicians will present papers on these subjects. This year's Congress will be held with special reference to surgery of coronary diseases. But presentations on any other subject in the field of diseases of the chest will also be accepted. The presentations will be followed by free discussions. The official languages for the Congress are: English, French, Spanish, and German.

As for more detailed information and inscription, please write to the secretariat of the Congress: Fourth International Congress of the American College of Chest Physicians, Köln-Deutz, Germany, Messeplatz.

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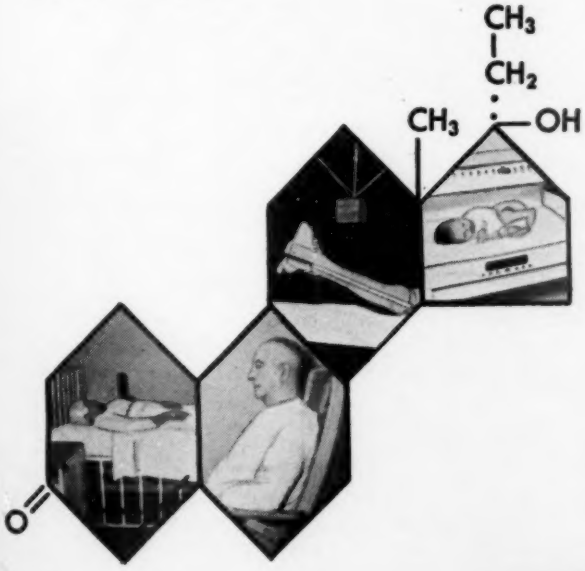

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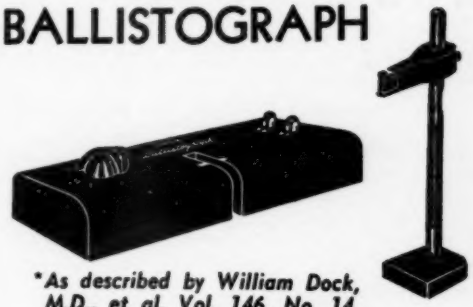

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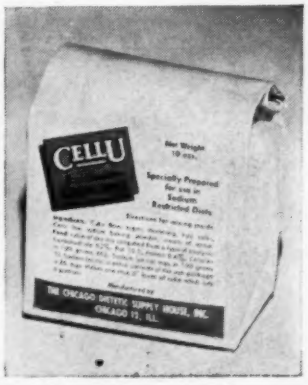

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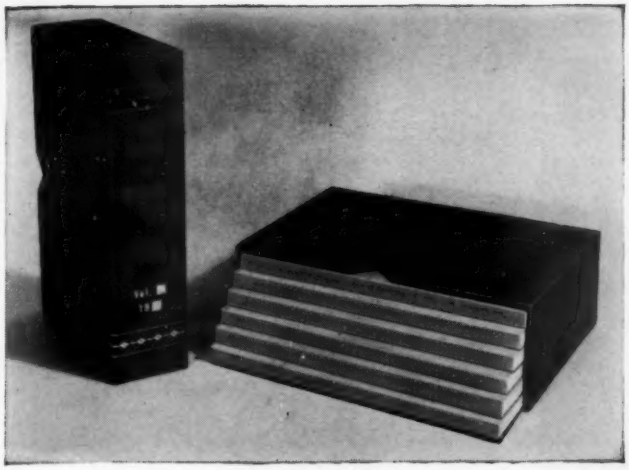

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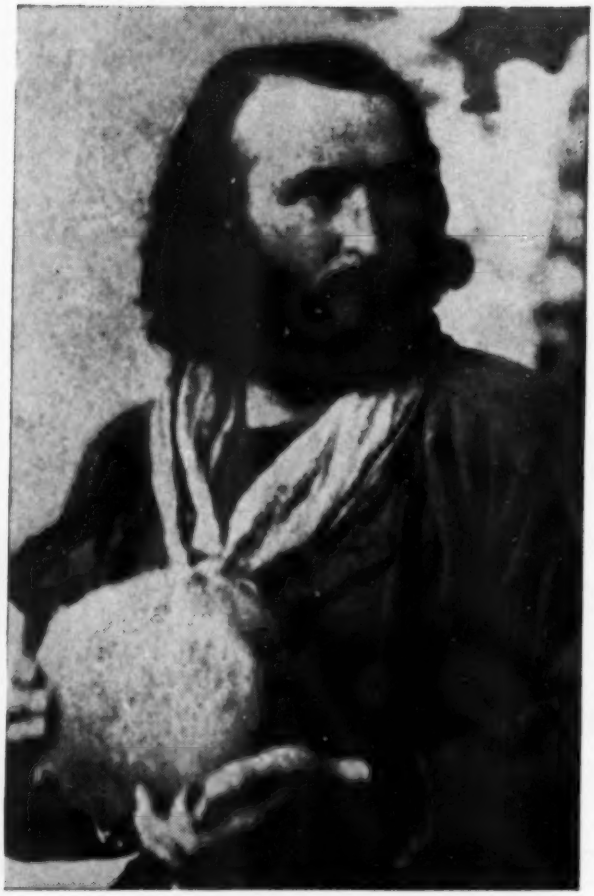

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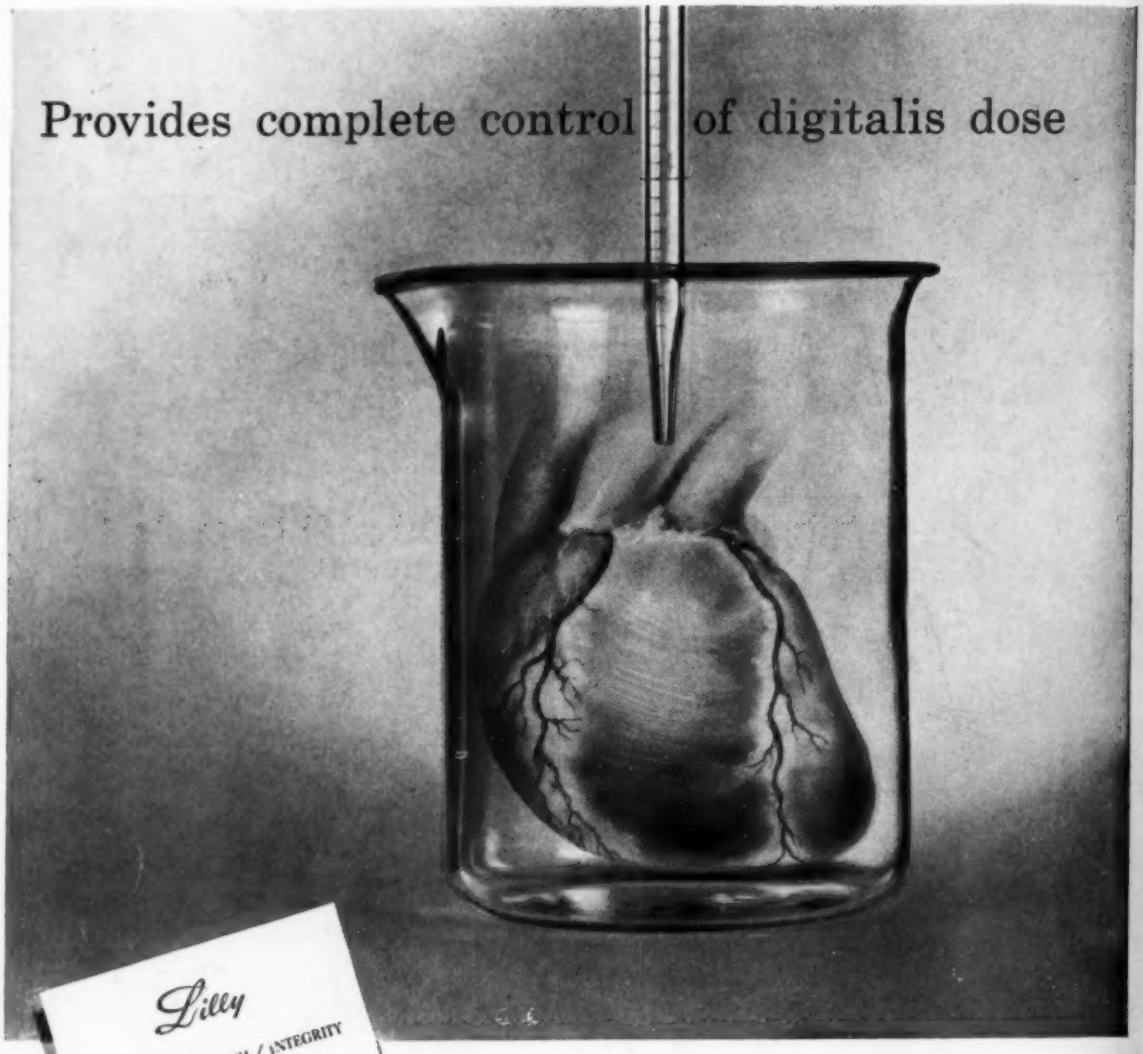

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